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ON TELANGIECTASIS CIRCUMSCRIPTA UNIVERSALIS.

By WILLIAM OSLER, M. D.

For many years I have been interested in the nævi and small telangiectic spots which one sees so frequently in the routine examination of patients. Their increase as age advances, their peculiar distribution, their temporary character in young persons, the association with cirrhosis of the liver, the possible association with internal carcinoma, the occasional eruptive-like outbreak in jaundice, the remarkable hereditary form associated with epistaxis (of which I have reported three cases)[1] the presence of the spider-nævi in scleroderma, and their occurrence in the scar of X-ray burns—these are points upon which I have dwelt over and over again in the routine work of the wards. On January 21, 1906, while I was taking one of Dr. Barker's ward classes, I found a patient whose case is here described, and I saw immediately that it was a form of generalized telangiectasis which I had never met with before. The case belongs to an excessively rare form of the disease of which only some fifteen or twenty cases are on record, and Dr. Barker has very kindly allowed me to report it. The history may be given in full:

W. J. H., age 39.—Patient complains of pain in the right side of abdomen.

F. H.—Family history is negative. The parents are living and well. He has no brothers nor sisters. His parents deny emphatically any joint or skin trouble in the family, but the mother had urticaria when young.

P. H.—Patient has not had any infectious disease. He has not had tonsillitis. He had attacks of "grippe" for several consecutive winters. The first, during an epidemic in Paris in 1889, was severe. In 1893-94 he had "pleurisy" though from his description one would suspect it to be lumbago. The pain was in the lumbar region, chiefly in the right side, very severe, relieved by hot packs and turpentine, in many ways resembling his present pain except that the former attack has been entirely in the back. He has believed his back to be weak and has taken gymnastic exercises. He has always been of neurotic temperament, and after 3½ years of hard work with much privation as an artist in Paris he broke down in 1893 with "nervous prostration," and for six years could work only intermittently. He thinks he has never entirely recovered.

Head.—He has had attacks of giddiness about once a week for past three years. He has considerable astigmatism which causes severe headaches relieved by glasses. Has never had any flashes of light; has never fallen; never has vertigo; no ear trouble.

Respiratory system.—No chronic cough, bronchitis, or haemoptysis.

Cardio-vascular.—During the past three months he has been rather short of breath after meals and on running up stairs. No pain around the heart.

Renal.—Not any oedema of ankles or of face. No blood in urine before present illness. The urine has been examined several times in the past few years and always found normal. No increase in frequency.

Gastro-intestinal.—No symptoms on the part of these organs; bowels always regular.

Habits.—Until six years ago he was an excessive smoker; since then moderate. Formerly a moderate drinker; now abstemious. Denies all venereal trouble.

Skin.—The skin condition has evidently not attracted much attention. The patient states he had noticed the purplish mottling only for the past ten years. His mother is sure that his

[1] Johns Hopkins Hospital Bulletin, 1901, Vol. XII, 333.

skin was normal when a baby and during youth and she has noticed the present condition only during the past two years. The patient says the mottling has become more intense during this time. At no time has it entirely disappeared, although more intensely colored during cold weather than in summer. During the summer of 1889 he had an attack of hives, and he gives an indefinite history of several attacks since. In November, 1905, he began to have epistaxis which has recently recurred without apparent cause and lasting about five minutes. During his attack of "nervous prostration" he was troubled with hæmorrhoids and was operated on. He has never noticed any tendency to prolonged bleeding from slight cuts. Has never had hæmoptysis. He denies absolutely any attacks of joint pains, colic, vomiting, or diarrhœa.

During the past month the patient's feet have bothered him by intense itching, so severe as to cause him to rise at night and apply a lotion. During the past two weeks patient has undergone a great deal of mental and physical exhaustion in connection with an art exhibition.

P. I. —Came on suddenly at six o'clock in the morning of January 20 (yesterday), with the passage of about a pint of bloody urine (dark red), followed by pains in the right abdomen "just below the last rib on the right side." Gradually the pain became extreme, and in ten minutes was at its height, causing the patient to double up and roll about in agony. The pain remained localized and did not radiate to the thighs or shoulder, nor was it paroxysmal. The pain lasted about twenty minutes and then gradually ceased, the patient breaking into a free perspiration. In half an hour after the onset of the pain he felt all right again, arose from bed and went about his days work (mounting and selling pictures). Last night he retired at 11.30. During the day patient passed his water three times and while it appeared dark, he did not notice that it contained blood. Last night the patient slept fairly well and did not get up to urinate. About six o'clock this morning (January 21), patient had a peculiar sensation in right abdomen, and a "presentiment of another attack." He arose from bed and passed another pint of brick-red urine, during which passage there was no pain, but immediately afterward pain came on gradually and in five minutes was extreme. A doctor was summoned who diagnosed the case appendicitis. The pain was agonizing and this time lasted five hours (until morphine was given sufficient to relieve it), and was accompanied by fever. The patient does not remember that he was short of breath during the attacks. In the first attack he had considerable nausea but was unable to vomit. In the second attack he vomited freely, especially after taking morphia. He has had no diarrhœa with the attacks. His appetite is good and he says it always has been.

Since the onset of his trouble the patient has noticed a prickly sensation in the end of his penis on urination.

Patient's mother says that the urine passed in the first attack contained blood definitely, but she is quite sure there was no blood in the urine passed before the attack on the second morning.

Fairly well nourished man. Face rather flushed: keeps eyes closed: pupils rather wide. *Pulse* 68, with occasional slight irregularity. Vessels well felt but not sclerotic.

Thorax.—Is symmetrical, except that right side is slightly fuller than left, and sternum deviates slightly to left. There is a well-marked lateral curve to right in mid-dorsal region. Movements of chest are equal. Lungs clear in front.

Heart.—Apex impulse visible and palpable in fifth interspace about in mammillary line, 9.3 cm. from median line and dulness extends 3.7 cm. to right. Impulse is of moderate intensity. First sound at apex slightly prolonged, suggesting a systolic murmur. Second sound clear. Sounds are clear elsewhere. Second left or pulmonic, a little louder than second right. During examination a number of patches of urticaria have appeared. There is fairly well-marked dermatographia and where he has been blue-penciled there is urticaria, which is appearing also as wheals bordered by a wide blush over places marked for dermatographia. *Abdomen* is natural. *Liver* is not palpable or enlarged. *Spleen* is readily palpable, falling, with patient on right side, 3-4 cm. below costal margin. The tenderness previously present has now disappeared.

R. B. C.....5,320,000
W. B. C.....11,900
Hb. .....106 (corrected).
Coagulation time = 4 mins. (slide method).

Fresh blood cells.—Appear of good color and uniform in size and shape. Not much tendency to rouleaux formation nor crenation. Leucocytes rather numerous but no marked leucocytosis.

On January 24 I made the following note: The skin presents a very remarkable appearance. On the face there are a few spots like acne rosacea. The skin of the neck is clear. Over the trunk and the extremities are numerous dark red spots looking exactly like a fresh purpura. Their distribution is very well shown in the accompanying photographs. They are very thickly set over the chest and back and on the flexor surfaces of the forearms and inner aspects of the arms (Figs. 2 and 3). The spots vary in size from two to six millimeters in diameter, and often coalesce to form large blotches. While of a dark purplish tint, as a rule, they can be changed by rubbing to a vivid red. Everywhere on palpation the spots disappear completely, leaving a slight brownish stain. They are not raised and the color is uniform. There are no individual blood vessels seen. It is evidently a capillary dilatation. It is remarkable the difference in appearance after friction of the spots on the arm. They become of a bright red color, return instantly after pressure, while the other spots are of a dark livid hue and the blood returns very slowly. The condition of the hands and feet is very remarkable. As seen in Fig. 1, the fingers are cyanotic and look like the picture of Reynaud's disease. The soles and margins of the feet and the toes have the same deep purple color. Factitious urticaria is readily produced, as is well shown in Fig. 4. The patient remained in hospital until March 9. He was on a modified Weir Mitchell treatment and did remarkably well. Dr. Bordley reported that he had a slight choroiditis in the right eye. During his stay in the hospital the patient had several severe attacks of abdominal pain, which was relieved by acupuncture. I saw the patient again an January 2, 1907. The skin was in practically the same condition. Though still somewhat nervous, he had kept pretty well and was able to attend to his work. The best account I have found of this condition is in *La Practique Dermatologique*, T. IV, by Brocq, under the title of Primitive Generalized Telangiectasis. Very few of the reported cases have had anything like the same extensive distribution as in the one here described. In one reported by Vidal[2] he calls it *Télangiectasie accidentale symétrique et généralisée*—a female, aged 31, nervous and hysterical; at the age of fourteen noticed the red spots appearing under aspects of the forearms. They extended gradually

[2] Bull. d. la Soc. Méd. des Hôpitaux, 1880-81, page 186.

FIG. 1.

FIG. 3.

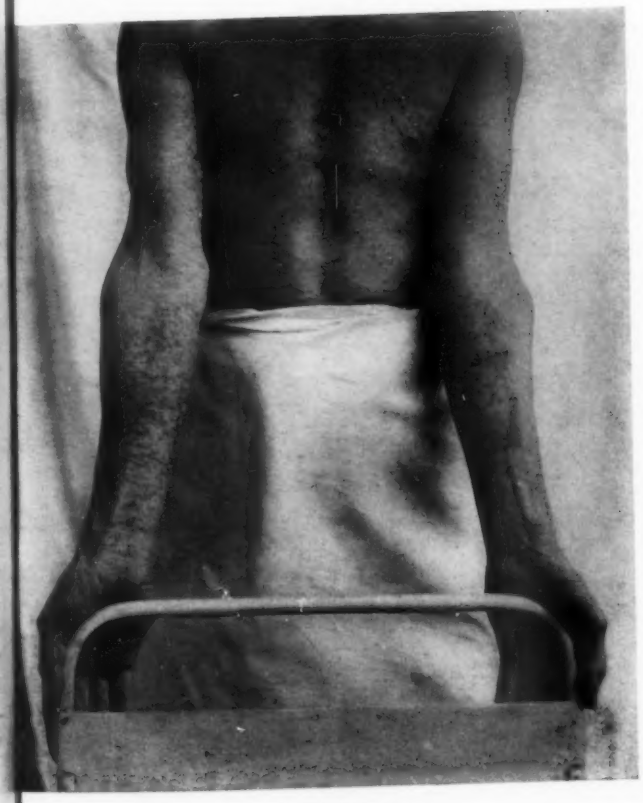


FIG. 2.

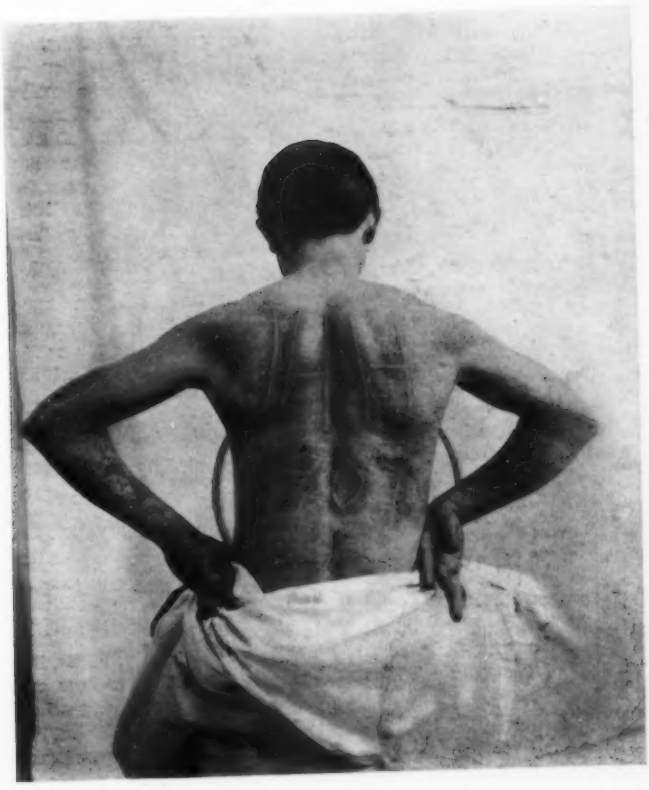


FIG. 4.

and appeared symmetrically on the arms, chest, neck, fingers, the backs of the hands, and the lumbar and dorsal regions. Before they came out she was very much troubled with a chronic urticaria, and she had a very marked hyperæsthesia of the skin. Levi [3] has reported two cases, the second one, a woman, aged 33, had only 35 spots in all, which had appeared in different parts of the body between 1897 and 1900. In his first case, a woman aged 70, the spots were much more extensive, and with a distribution very much as shown in the photographs here given, but there was not the extreme involvement of the feet and hands.[4]

One or two points about this case require comment. From the statement of the mother there can be little question that this is an acquired, not congenital form. Everywhere it is the capillaries, not the small venules that are involved. The appearance of the back and of the arms is not unlike that seen in the most extreme grade of vasomotor mottling. The patient, Juliet D., medical number, 16254, who was in the ward G in October, 1903, had an appearance of the back and feet very similar to this patient. She was also extremely neurotic, but under treatment the mottling entirely disappeared. This is a permanent dilatation of the capillaries of localized areas. The condition of the hands and feet suggests the local asphyxia of Reynaud's disease. The skin of the soles of the feet and the toes was quite purple. The color could be changed to a vivid red by friction. In the interval of a year, which elapsed after I first saw him, no change had occurred, so that it is evidently a state of permanent ectasia of the blood vessels of the skin. Dermatographia, common enough in conditions of vasomotor instability, is seen in a marked degree in many cases of neurasthenia. Two other symptoms are of special interest in the case. The recurring attacks of colic, for which no cause could be found, may have been associated with a gastro-intestinal urticaria, that is a localized area of infiltration of the gastro-intestinal wall, such as has been shown to be responsible for the colic in the so-called Hennoch's purpura. The hæmaturia may be a form of so-called renal epistaxis, such as is met with sometimes in Reynaud's disease. Bleeding is a common event in the remarkable generalized telangiectasis of the hereditary form, and some of the cases have been described as hæmophilia. Since the report of the cases in the *Bulletin*, already referred to, I have found another family. The bleeding is usually from the nose, but it may be from the lips or mouth, only rarely from the spots on the skin.

[3] Gaz. Hebdom., 1901, p. 13.
[4] Presse Médicale, 1896.

POSITION OF THE HEART IN PERICARDITIS WITH EFFUSION.

By W. J. Calvert, M. D.,

University of Missouri, Columbia, Missouri.

The position of the heart in pericardial effusions is of practical importance in tapping the sac. Consequently observers have, from time to time, attempted to determine its position and most naturally have arrived at various conclusions. Skoda (1) and his followers think the heart is displaced downward and backward. Schaposchnikoff (2) and others say the heart is pushed upward, anteriorly and may be in contact with the anterior thoracic wall. While a third group (3) contend that the heart remains in its normal position.

This difference in opinion indicates that the heart in pericarditis with effusion occupies various positions, which must be determined by physical factors or laws. These factors change or vary with the changing conditions within the pericardium. If these physical factors or laws can be determined and if clinical observations can be interpreted in terms of these laws, the position of the heart in a given case of pericarditis ought to be fairly accurately determined.

The aim then of this article is to determine, as far as possible, the physical factors which govern the position of the heart in pericarditis.

Material.—The material for this article was obtained through the kindness of Dr. C. M. Jackson, to whom I wish to express my thanks. The material consists of the cadavers of two individuals who, at the time of death, had pericardial effusions. The cadavers were hardened by an intra-arterial injection of formalin and sectioned transversely. From these sections drawings and measurements were taken by means of which the organs have been projected. This material is designated as Cases I and II. The two cases were treated in exactly the same manner, consequently any changes in the cadavers due to manipulation should be the same in each.

Three pictures showing the position of the heart in pericarditis with effusion are reproduced from the Anatome Topographica, by Nicolao Pirogoff, published from 1852 to 1858. I wish to express my thanks to the library of the Surgeon-General's office for the use of this atlas.

Case I.—The cadaver is that of a fairly well built male negro, aged thirty. A left sided pleurisy with a very small effusion, adherent right pleurisy and pericarditis with effusion were observed.

The position of the pericardium is shown in Figs. 1, 2 and 3 It extends from the second to the seventh rib in the left mammary line, and the seventh rib in the right sternal line; the lungs have been displaced outward, and the diaphragm downward.

All of the chambers of the heart are collapsed (Figs. 1 and 3). The heart is against the posterior wall of the pericardium, separated by fluid from the anterior wall. In Fig. 2 (anterior projection) the upper border of the heart is at the top of the third rib; apex, six centimeters from the mid-sternal line; the right

border, three and three-fourths centimeters to right of mid-sternal line; and the lower border on a level with the seventh rib. The apex is displaced backward and slightly to the right (Fig. 3). The downward displacement of the diaphragm has not been followed by the heart. There are no adhesions between the heart and pericardium.

The venae cavae are collapsed as shown in Fig. 1, *f*, and *i*. The vessels are collapsed from their entrance into the pericardium to the heart. Their lumen may be represented by a Y-line. Outside the pericardium the veins are congested.

The lumen of the aorta is obliterated, flattened in the anterior-posterior diameter.

The pulmonary arteries and veins are congested outside the pericardial sac.

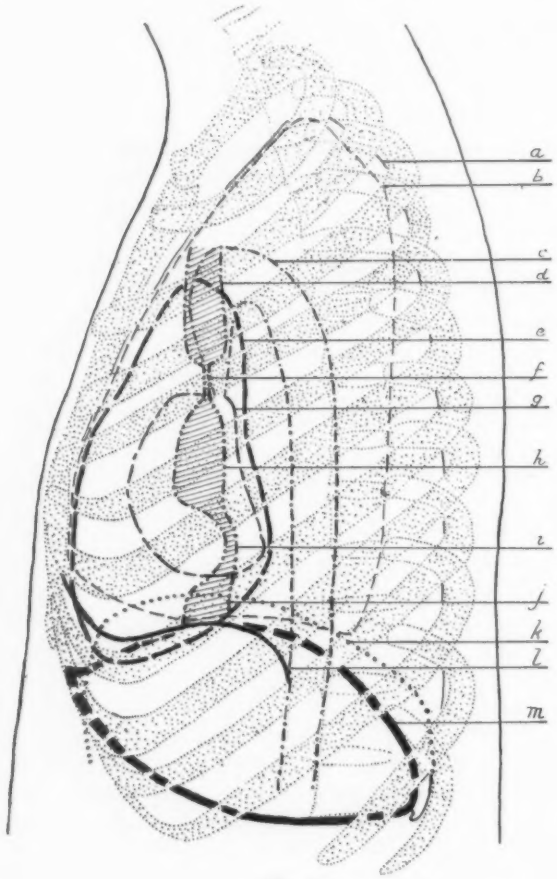


FIG. 1.

CASE II.—Cadaver is that of a male negro, aged twenty-nine, well formed and fairly well nourished. Examination revealed tuberculous consolidation, with multiple small cavity formations in the left lung, and pericarditis with effusion.

The pericardium extends from the middle of the first interspace (mid-sternal line) sixteen and one-half centimeters downward, almost to end of the sternum; eight and one-half centimeters to left and six and one-half centimeters to right of mid-sternal line. Its relationship to the anterior thoracic wall and the lungs is well shown in Figs. 4, 5, 6, and 7. The lower wall of the pericardium is pushed downward with the diaphragm (Fig. 4).

All of the chambers of the heart are distended with blood (Fig. 6). The position of the heart in an anterior-posterior diameter and its relationship to the anterior thoracic wall and lungs are sufficiently well shown in Figs. 5, 6 and 7, to dispense with further description. The projection of the heart (Fig. 4) extends from the upper border of the third rib to the articulation of the seventh costal cartilage with the sternum, six and three-fourths centimeters to the left and five centimeters to the right of the mid-sternal line. The heart is in a relatively normal position. The lower wall of the pericardium has been displaced downward leaving the heart absolutely free to support itself in the pericardium (Fig. 4). There are no adhesions between the heart and pericardium.

The venae cavae are distended throughout and continuous with the distended right auricle (Figs. 4, 5 and 7).

The aorta is distended (Figs. 4 and 5).

The pulmonary arteries and veins are distended (Fig. 5).

COMPARISON OF THE TWO CASES.

FACTORS IN COMMON.

1. The pericardial sac is distended relatively the same in the two cases; neither of them are large effusions.
2. Pulmonary congestion in each.
3. Systemic venous congestion in each.
4. In each the heart is free in the pericardium, consequently uninfluenced by adhesions.
5. In each the projection of the heart on the anterior thoracic wall shows a relatively normal position.
6. In each the heart was completely surrounded by fluid, consequently the buoyant force was relatively equal in the two cases.
7. In each case the heart swings in the sac absolutely independent of the lower wall of the pericardium and diaphragm.
8. Gravity must have been equal in the two cases.
9. A dorsal position of the two patients at the time of death may be assumed. When the cadavers were injected and hardened, both were in the dorsal position.

DIFFERENCE IN FACTORS.

Case I. Heart chamber collapsed.
Case II. Heart chamber distended.
Case I. Heart displaced backward not in contact with anterior thoracic wall.
Case II. Heart immediately behind the anterior thoracic wall, practically in its normal position.
Case I. Venæ cavæ within pericardium collapsed.
Case II. Venæ cavæ within pericardium distended.
Case I. Left auricle collapsed.
Case II. Left auricle distended.

SUMMARY.

Case I. Heart and vessels collapsed. Heart against posterior wall of pericardium, in posterior position.

Case II. Heart and vessels distended, in relatively normal position.

A casual consideration of the two cases shows that:

1. The heart may or may not be in its normal position.
2. That it is not displaced upward to the anterior.

3. That it does not sink downward when the support of the diaphragm is withdrawn.

4. When not in the normal position the heart is displaced backward against the posterior pericardial wall.

5. The distension of the heart's chambers vary under varying conditions.

congestion. While compensation is maintained a normal quantity of blood is delivered to the auricle which must remain relatively normal in size. The auricular pressure must increase as the pericardial increases; and at no time can it be less, else the auricle must collapse, when death would follow. As compensation fails and less blood is deliv-

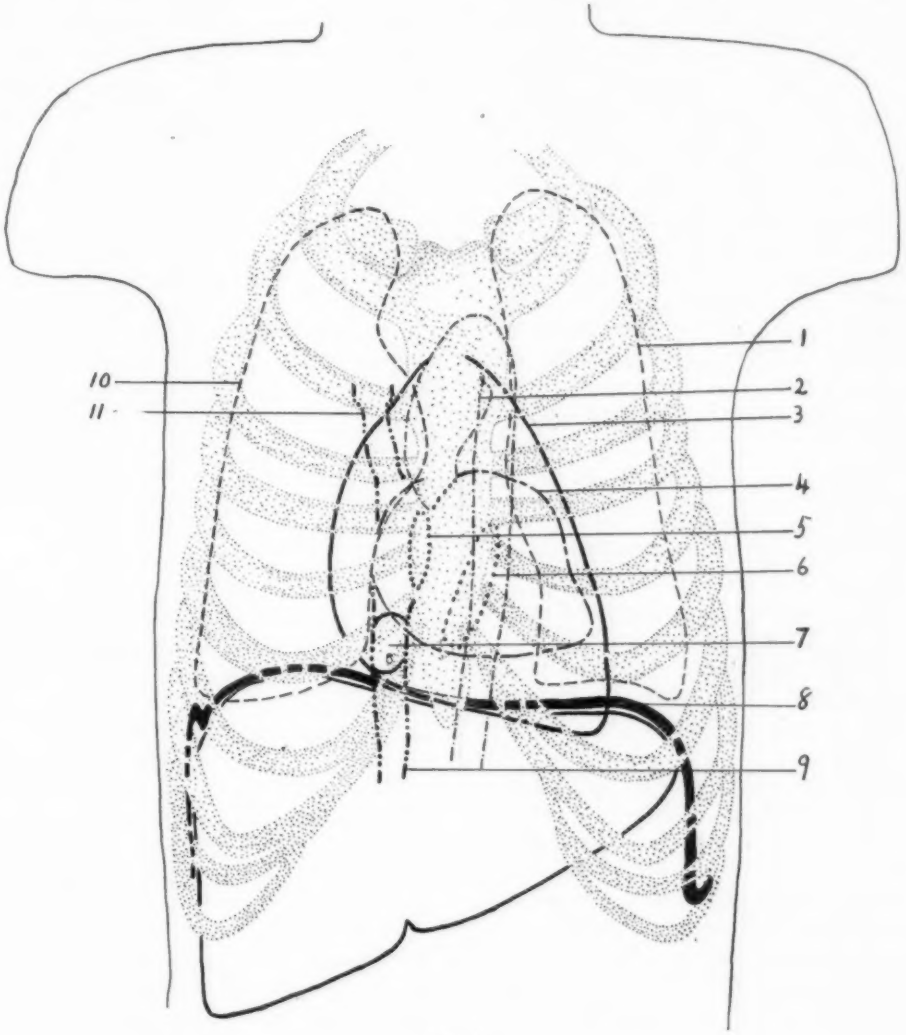


FIG. 2.

The circulatory changes herein considered have been studied in the light of physiological and clinical data. In this consideration the heart is supposed to be free from valvular lesions. The order of description is: left auricle, right ventricle, right auricle. The left ventricle is considered in the paragraphs on compensation, amount of blood delivered to the ventricles, pulse rate, etc.

Left auricle.—When the pericardial pressure first affects the auricle, the auricle must be compressed to a point where the intra-auricular pressure equals the pericardial, or the auricular pressure is increased and must cause pulmonary

ered to the right heart, the auricle diminishes in size, until practically no blood reaches it, when it is collapsed as in case I.

The pulmonic congestion must vary with the pressure in the left auricle, consequently must continue throughout the course of the disease.

Right ventricle.—The right ventricle must pump its blood into a high pressure pulmonic circulation. To accomplish this increased work a certain amount of hypertrophy and perhaps some dilatation are necessary.

Right auricle.—The right auricle and venæ cavæ within

the pericardium are subjected to the pericardial pressure and must be compressed, this obstructs the flow of blood from the superior and inferior cava, causing a congestion and rise of blood-pressure within the systemic veins. This increased venous pressure must compensate the effect of the pericardial pressure on the auricle and venæ cavæ within the pericardium. So long as this compensation continues a relatively normal quantity of blood flows to the right auricle, thence to the right ventricle. But when this compensatory congestion begins to fail the lumen of the vena cava within the pericardium becomes progressively smaller, consequently less and less blood flows to the right auricle, thence to the right ventricle. The quantity of blood entering the right auricle must determine the quantity of blood in the heart. The pressure within the right auricle must be like that in the left, slightly greater than that in the pericardium, consequently the pressure in the systemic veins must be above that in the pericardium, and must offer an opportunity to measure the pericardial pressure. The right auricle, like the left, is gradually compressed by the effusion, until collapsed, all the while maintaining a high auricular pressure.

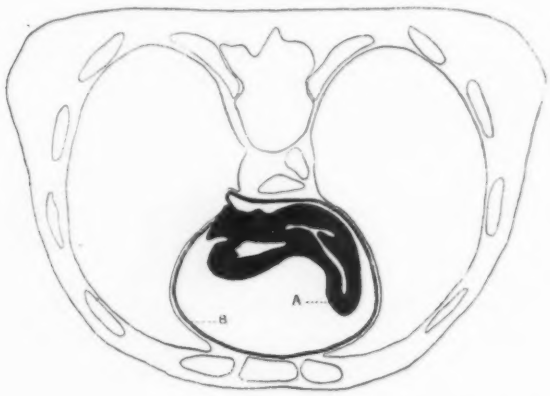


FIG. 3.

Compensation.—To complete the cardiac circulation the blood must enter the pericardium twice, consequently the pericardial pressure must be compensated twice:

1. The left auricle is compensated by the right ventricle.

2. The right auricle, by the increased pressure in the systemic veins due to congestion.

As the two mechanisms for compensation are independent each may remain competent while the other fails. If the right ventricle fails, while the venous compensation is competent, the right auricle would continue to pump blood into the right ventricle, which would dilate, but would force little or no blood to the left heart, which must collapse. Traube[3] describes a case illustrating this point. He says: "The heart is very small, and peculiar in that the diameter of the base is greater than the longitudinal diameter. The contents are fluid and large in quantity on the right side."

If the venous compensation fails little or no blood enters the right heart, a like amount, the left. Consequently the heart must gradually collapse, because there is not enough blood to distend it. This is shown in case I.

Blood delivered to ventricles.—The amount of blood delivered to the ventricles must depend on the degree of compensation. In the early stages of the disease the auricles are perfectly compensated with perhaps a slight margin of reserve force. During this stage a normal amount of blood should be delivered to the ventricles. (On account of the increased auricular pressure a slightly increased amount of blood may flow into the ventricles.) As compensation fails a diminished amount of blood is delivered to the right auricle, consequently to the ventricles.

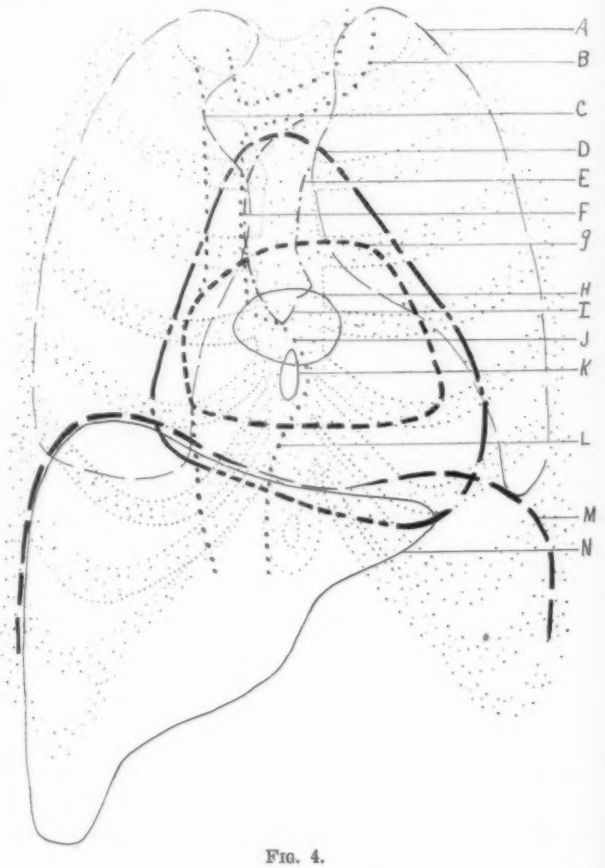


FIG. 4.

Pulse rate.—The pulse rate is usually increased throughout the course of the disease and is due to different causes in the several stages of the disease.

1. In the beginning the ordinary febrile pulse must be present.

2. The distension of the large veins is a sufficient mechanical stimulus to increase the heart's action.

3. As the amount of blood entering the heart diminishes a low arterial tension develops. This the heart attempts to compensate by increased activity.

4. If the venous pressure becomes very high a reflex diminution in pulse rate may follow.

5. If the vagus is involved a slow pulse rate may develop.

Blood-pressure.—When the pericardial pressure is first developed, the high pressure in the auricles may for a time deliver to the ventricles more than the normal amount of blood which, with the increased rate of the heart, may give an increased arterial tension. If this is true, it is only temporary. When compensation has developed, a relatively normal blood-pressure should be maintained. Hensen[8] reports a normal blood-pressure in two cases with large effusions. As compensation fails the arterial pressure falls; or the arterial tension falls as the pericardial pressure rises. This decrease in arterial tension was first experimentally demonstrated by François Frank.[1]

Coronary circulation.—The coronary vessels are subjected to the direct pericardial pressure, and the flow in the veins is obstructed by the increased auricular pressure. These factors must seriously interfere with the coronary circulation, more especially in the later stages of the disease, when the arterial tension becomes much diminished. There is no compensation for this impediment to the coronary circulation. The resulting anæmia may in large measure account for the myocardial changes noted.

Work of the ventricles.—While compensation is maintained the pulmonic blood-pressure is higher than normal, the systemic blood-pressure normal. Consequently the right ventricle has an increased amount of work to do, the left a normal amount. As the disease progresses and compensation fails the pulmonic blood-pressure is increased, the systemic decreased. The right ventricle has an increasing, the left a decreasing resistance to overcome.

Position of the heart.—In the cited cases the heart is shown in positions ranging from relatively normal to that shown in Case I, or completely collapsed and against the posterior wall of the pericardium. In each of the two cases described the heart is supported by the large vessels, in one it is collapsed, in the other, distended; consequently the degree of distension of the heart must determine its position in the pericardial sac; or the position of the heart depends on the cardiac hydraulic pressure acting against the attachments of the heart, and the amount of blood within the heart and that portion of the large vessels within the pericardium. The distension of the heart depends on the amount of blood delivered to the right auricle; this amount of blood depends on the compensatory venous congestion; and the latter depends on the relationship between the intravenous and pericardial pressures. So long as compensation is maintained the degree of pericardial pressure is not important; but as compensation fails the quantity of blood delivered to the right auricle varies inversely as the pericardial pressure.

In the standing position the greater portion of the weight of the heart and its blood is supported by the aortic and pulmonic arteries. The distended auricles fastened behind to the pulmonary veins and on the right above and below to the venæ cavæ, act as a brace or prop to keep the heart from falling backward on account of gravity. In the dorsal position the weight of the heart is transmitted through the distended auricles to the large veins, thence to the mediastinal tissues and the vertebral column. Here the aorta and pulmonary artery, attached anteriorly to the base, act in a measure as a prop to prevent side to side movement of the heart.

During the stage of compensation the quantity of blood in the heart remains fairly constant, consequently the position of the heart must remain as constant. (If the heart was in a normal position at time of onset of effusion it must remain in relatively a normal position while compensation is maintained.) But as compensation fails the quantity of blood delivered to the heart must gradually diminish, allowing both the auricles and ventricles to gradually diminish in size. This process continues until the heart is completely collapsed. The auricular brace above described becomes shorter, which allows the heart to swing backwards on the aorta and pulmonic artery as a hinge. The apex is displaced backward. The shortening in the long diameter of the heart probably compensates the downward displacement of the apex, due to its partial revolution about the aorta. Thus the apex is displaced backward and slightly to the right, but not downward or to the left.

During the course of the disease the heart occupies positions varying from normal to that shown in Case I, which is the greatest possible displacement of a normal heart by pericardial effusion.

To determine the pericardial pressure, or the state or stage of the compensatory congestion, is the surest method of obtaining reliable information concerning the position of the heart. This pressure is produced by:

1. The strength of the pericardial walls. Naturally, this factor must vary in each case and must determine the size of the effusion. If the sac can resist a pressure greater than that in the systemic veins, the sac will not dilate and the veins will be closed. If the sac is weak, it will easily dilate and allow the accumulation of a large effusion. In the former condition a high pressure may be developed early; in the latter, late or never.

2. The force under which the fluid enters the sac. Here the question of transudates, exudates, and osmosis have to be considered. They lead to no definite conclusions. That considerable pressure can be developed in the pericardium is certain.

The degree of pericardial pressure may be approximated by:

1. Shape of pericardial dulness. When pressure is high dulness must extend high, first or second interspace, and be triangular. When pressure is low the area of dulness is more quadrangular (Romberg, p. 370), and the movability of the dulness with change of position should be greater. The amount of fluid is of secondary importance.

2. The degree of venous congestion must be an index to the pericardial pressure.

3. Arterial tension should be low when pericardial pressure is high and should decrease as pericardial pressure increases.

Pulsus paradoxus indicates a pericardial pressure relatively equal to the maximum venous pressure.

4. The pulmonic symptoms should be more pronounced under a high than under a low pericardial pressure.

Support of the heart.—Regarding the normal support of the heart, the general opinion is that it is supported by the large vessels, pericardium, lungs, and the central tendon of the diaphragm. The two cases at hand show beyond a doubt that the heart is supported without the aid of the pericardium, lungs, or the central tendon of the diaphragm. Here it is natural to ask can the heart be supported under normal conditions without the aid of these structures?

It has been shown that while compensation continues the heart remains in its normal position and receives its normal amount of blood. During this period the pericardial pressure may vary through quite a wide range without changing the conditions of the heart. It is natural to suppose that compensation is an attempt of nature to maintain as long as possible and as near as possible the normal results of physiological activity. Consequently the increased cardiac and venous pressures are increased after and in proportion to the rise in pericardial pressure and counterbalance the pericardial pressure without taking any part in the support of the heart, in either of the two cases under consideration; and that the normal pressures and quantity of blood remain in the heart in addition to or above the pressure caused by the rise of pressure in the pericardium.

This leaves the normal cardiac pressure and amount of blood in the heart to support the heart in the two cases under consideration and permits the subtraction of the pericardial pressure and a like amount from the cardiac pressure without materially changing the position of the heart. This reproduces the normal condition (save the presence of the fluid) with the heart supporting itself without the aid of the pericardium, lungs, or diaphragm. Now suppose the pericardial fluid is withdrawn without changing the pressures, the walls of the pericardium come in contact. It is reasonable to suppose the heart continues to support itself without the aid of the neighboring structures.

Clinically it is well known that the heart moves a number of centimeters to the right or left as the case may be, consequently the support of the lung and pericardium cannot be very substantial.

If this be true the heart is independent, save in a most general way, of the pericardium, lungs, and diaphragm, and swings free in the pericardium to be influenced by a most delicate mechanism, namely the flow of blood to the heart, the cardiac pressure, and all in the thoracic cavity in which a slightly positive to a slightly negative pressure is maintained.

SUMMARY.

1. The position of the heart in pericarditis depends on the size of the heart.

2. The size of the heart depends on the degree of distension, or on the amount of blood within the heart.

3. The amount of blood within the heart depends on the stage of compensation.

4. While compensation is maintained the heart contains a relatively normal amount of blood and is in a relatively normal position.

5. When compensation fails the amount of blood delivered to the heart is diminished, the heart becomes smaller and smaller until collapsed. The size of the heart varies inversely as the pericardial pressure. During this change the heart successively occupies positions varying from normal to that shown in Case I, or against the posterior wall of the pericardium.

6. The apex is in a normal position or displaced backward and slightly to the right; otherwise unchanged.

7. When the pericardial pressure is high, or when compensation is failing the signs should be triangular area of dulness, high pulse rate, perhaps pulsus paradoxus, low arterial tension, marked systemic venous congestion, and pronounced pulmonary symptoms.

LITERATURE.

1. Skoda: The heart sinks backward and downward. Quoted by Thayer. Johns Hopkins Hospital Bulletin, 1904. Vol. 15, p. 149.

Ewart: Apex beat unaltered or even lowered. Osler's Text-Book, p. 780.

Friedreich, Riegel, and Bauer: The heart is in a posterior position. Quoted by Zezschwitz. Munch. med. Wochenschrift, Vol. 48, p. 1348.

2. Schapozhnikoff, after relating his experiments and stating his conclusions, says: This tendency of the heart to remain near the anterior chest wall is due to the influence of its attachments to the basic vessels. If these vessels are ligated and the heart and its contents set free it will fall to the bottom of the pericardium. The heart does not sink by virtue of its specific gravity, as has been thought, but floats and is found to be above the pericardial fluid. Extract in the American Journal of the Medical Sciences, Vol. 131, p. 713, from Revue de Médecine, 1905, XXV, p. 789. This is a translation of the original in Russk. Arch. pat. klin. Med. 1. bakt., St. Petersburg, 1896, II, p. 75.

Frænkel: During a radical operation the heart projected into the opening, preventing the outflow of fluid, save during systolic contraction. Zur Lehre von der Perikarditis. Beiträge zur wissenschaftlichen Medicin und Chemie. Festschrift zu Salkowski. Berlin, 1904, p. 19.

Thayer, case II, says: "Friction rub—large heart near anterior thoracic wall—fluid posterior and to left." Johns Hopkins Hospital Bulletin, 1904, Vol. 15, p. 149.

Zezschwitz found the heart in an anterior position in one case. One of Pirogoff's cuts is reproduced on page 1351. Munch. med. Wochenschrift, Vol. 48, p. 1348.

3. Scott and Le Conte, in conclusion number four, say: "It is yet to be proved that the heart is pushed upward and backward in effusion." The American Journal of the Medical Sciences, Vol. 128, p. 447.

FIG. 5.

FIG. 6.

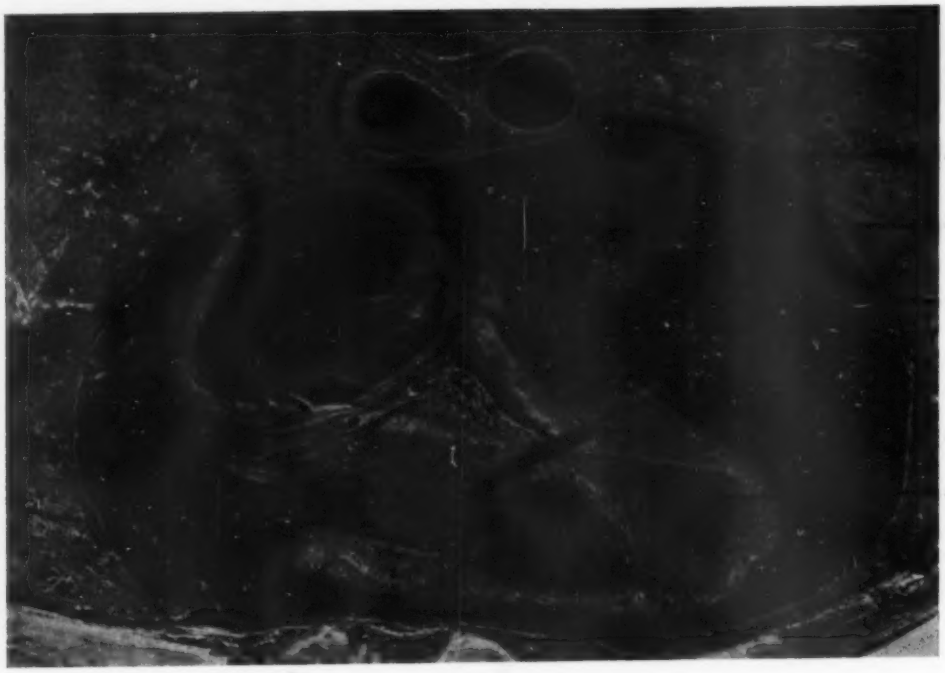


FIG. 7.

FIG. 8.

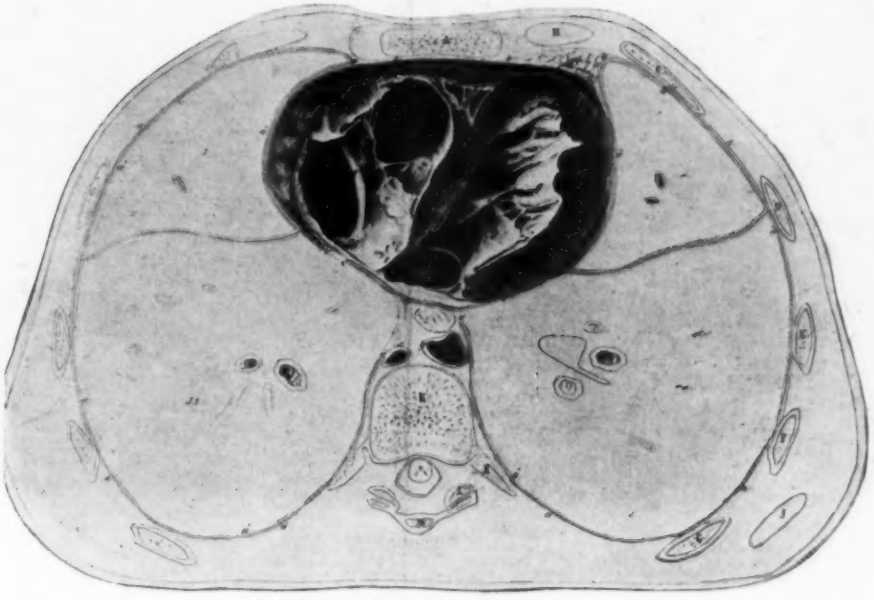


FIG. 9.

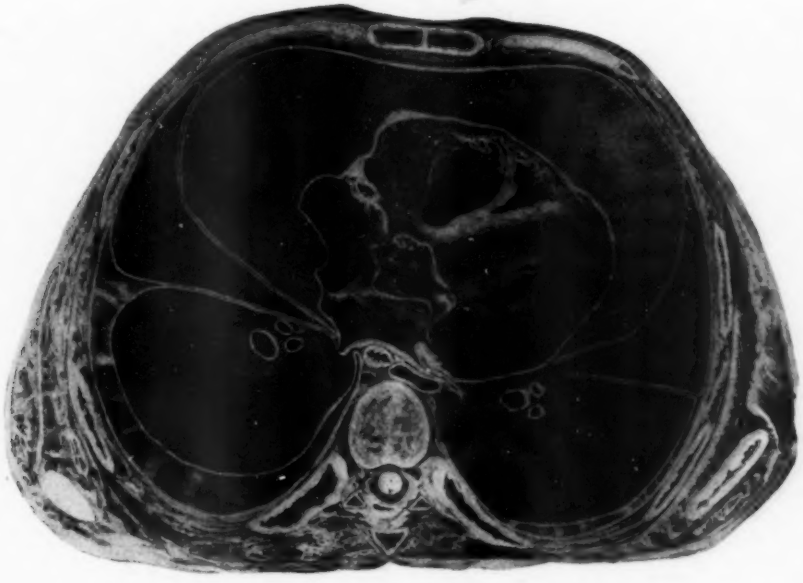


FIG. 10.

Romberg says that the heart rarely sinks in the fluid, and is separated from the anterior thoracic wall by a thick layer of fluid only in cases of very large effusions, p. 370; again, p. 375; the heart is seldom displaced to a marked degree. Lehrb. d. Krankh. d. Herzens, 1906.

Damsch says that the heart is in its normal position, but is not in contact with the anterior thoracic wall. Zeitschrift f. klin. Med., Bd. 38, p. 285.

Rendu and Ferrand say that the heart is retained in its normal position by the large vessels. Quoted by Thayer l. c.

Uslenghi says that the heart is in its normal position and not displaced downward. Semana Medica, Buenos Ayres, Vol IX, p. 286.

4. Warthin says that an early and persistent accentuation of the second pulmonic sound may be present. Osler, p. 780.

5. Gesammelte Beiträge, Bd. 3, p. 135.

6. Hensen, Deutsch. Arch. f. klin. Med., Bd. 67, p. 436.

7. François Frank, Gaz. hebd., 1877, No. 29.

DESCRIPTION OF FIGURES.

Pictures of Case I.

Fig. 1.—View of organs from left side. *a*, Parietal pleura; *b*, left lung; *c*, aorta; *d*, superior vena cava; *e*, pericardium; *f*, collapsed superior cava; *g*, heart; the heart is curved as shown in Fig. 3, so that this projection is much thicker than the heart really is; *h*, right auricle; *i*, collapsed inferior cava; *j*, inferior vena cava; *k*, right cupola of diaphragm; *l*, central tendon of diaphragm; *m*, left cupola of diaphragm.

Fig. 2.—Anterior projection of Case I. 1 and 10, lung borders; 2, aorta; 3, pericardial wall; 4, outline of collapsed heart; 5, tricuspid valve; 6, mitral valves; 7, collapsed portion of inferior cava; 8, diaphragm; 9, inferior cava; 11, collapsed portions of superior cava.

Fig. 3.—Transverse section of body showing flattened condition of the heart (*A*) and its relation to the pericardial wall (*B*).

Figures from Case II.

Fig. 4.—Anterior projection of Case II. *A*, lung border; *B*, jugular veins; *C*, superior cava; *D*, pericardial wall; *E*, aorta; *F*, superior cava; *G*, outline of the distended heart; *H*, mitral valve; *I*, aortic valve; *J*, right auricle; *K*, tricuspid valve; *L*, inferior cava; *M*, diaphragm; *N*, liver.

Fig. 5.—Transverse section of body just above the heart. *A*, pulmonary artery; *B*, superior vena cava; *C*, aorta; *D*, conus arteriosus; *E*, pericardium; *F*, pulmonary vein.

Fig. 6.—Transverse section through body showing the cavities of the heart and relationship of heart to pericardial wall. *A*, left auricle; *B*, pericardium; *C*, mitral valve; *D*, left ventricle; *E*, right auricle; *F*, right ventricle.

Fig. 7.—Transverse section of body showing lower portion of the pericardial sac and distended inferior vena cava. *A*, pericardium; *B*, lower portion of right auricle and inferior vena cava; *C*, coagulated effusion in pericardial cavity; *D*, lower portion of the left ventricle.

Figures Nos. 8, 9 and 10 are reproduced from Anatome Topographica, by Nicolao Pirogoff.

Fig. 8.—Shows a sagittal section of the body. The conus arteriosus, pulmonary valve, and a portion of the distended pulmonary artery well shown. The position of the heart in the pericardium is clear.

Fig. 9.—Transverse section of the body, under surface, the right heart is to left of picture. The cut shows a small pericardial effusion, the heart dilated and in a relatively normal position.

Fig. 10.—Transverse section of body, under surface of section shown, right heart to left of section. The heart is partially collapsed and separated from the anterior wall.

THE FETAL CIRCULATION THROUGH THE HEART.

A REVIEW OF THE MORE IMPORTANT THEORIES, TOGETHER WITH A PRELIMINARY REPORT ON PERSONAL FINDINGS.

By Augustus G. Pohlman, M. D.,

Junior Professor of Anatomy, Indiana University.

It would seem from the description in the various text books that the course of the blood through the fetal heart had been definitely settled. There are three major theories on the question which are reviewed in order of their general acceptance and inversely in the order of their appearance:

1. The first is the theory of Sabatier which appeared about the beginning of the 19th century. The blood entering the right auricle through the inferior cava was believed to cross (at least for the most part, Bichat's modification) through the foramen ovale to the left side. The more arterial blood in the left ventricle was expelled through the aorta, and in consequence of the origin of the large arteries to the head and front extremities, the more rapid growth, particularly of the head, was believed to be accounted for satisfactorily. A critical examination of this theory, which by the way, is the prevalent one at the present time, shows it to be physically impossible, morphologically inaccurate, and developmentally unnecessary. Physically impossible, because it would entail a distinct crossing of two currents under equal pressure within a distending chamber. Morphologically inaccurate, as Born pointed out, in that no such arrangement is found in the sauropsidian embryo, and still the head develops more rapidly, or even more strikingly in the human embryo where the leg buds, once they appear, develop more rapidly than the arm buds when on the Sabatier principle the reverse should be the case. Developmentally unnecessary in that a number of organs go rapidly from anlage to a relatively large size without being vascularized (kidney, testis). Nevertheless this theory is almost universally accepted and is pictured in practically all the textbooks on anatomy, embryology, and obstetrics.

2. The second theory was suggested by C. H. Wolff (1775),

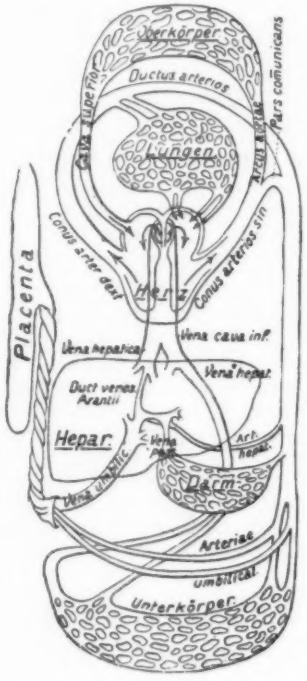
and although of earlier date, is a decided improvement on the foregoing. Wolff was able to appreciate a doubling of the orifice of the inferior cava or to put it in the words of Preyer (1884), "Es kann jetzt nicht mehr zweifelhaft sein, dass Casp. Friedr. Wolff Recht hatte, wenn er auf Grund seiner sehr sorgfältigen anatomischen Untersuchungen (1775) behauptete, der linke Vorhof erhalte gar kein Blut aus dem rechten Vorhof, sondern nur aus der unteren Hohlvene (und später den Lungenvenen), welche hinten an der Grenze beider Atrien, wie er fand, doppelt einmündet, so dass die linke obere Mündung nur dem linken, die rechte untere Mündung nur dem rechten Vorhof Blut zurführt. *Das Blut der beiden Vorhöfe kann sich also garnicht mischen*, wie auch Sabatier richtig betonte, und wie ich nach eigener Anschauung ebenfalls behaupten muss" (italics mine).

Preyer, however, still maintained that the head must receive a better quality of blood, although he believed the more arterial blood of the inferior cava to be divided equally to the right and left venticles. He argued that the pulmonary circulation was equal in quantity to that of the head and upper extremities and inasmuch as the lungs needed less oxygen, the return through the pulmonary veins must necessarily be purer than that of the vena cava superior. In consequence the blood in the left ventricle was more arterial than that in the right, and the result was similar to Sabatier's scheme, minus the disagreeable physical factor mentioned.

Preyer's findings were substantiated by his student, Ziegenspeck, in 1884. The latter presented an unique diagram, in which he represented the heart as two distinct hearts. After about twenty years (1905) Ziegenspeck has revived the theory once more and illustrated it by a figure which I have copied and which bears the title "Richtiges Schema des Placentarkreislaufs." It might be well to state that Ziegenspeck's article is detailed and carefully worked out, including the results of 36 injections into the umbilical vein of embryos.

Ziegenspeck really betters Preyer's work in that he eliminates the idea of a definite quality of blood and yet adheres to the quantity, believing therein to simplify the whole matter. The data furnished by Ziegenspeck might be questioned, because no matter how carefully the conditions are reproduced a dead animal is never the same that it is during life. The careful calibration of the various vessels is equally open to criticism because the amount of normal capillary resistance in the various vessels is probably different. There is at least no basis for assuming it to be the same.

Apart from this very general criticism, Ziegenspeck's figure itself is open for debate. Interpreted the figure means this: grant the contents of either ventricle to represent a volume equal to 5. Then inasmuch as the blood from the inferior cava is distributed equally to either ventricle, other return to the right auricle (vena cava superior, coronary sinus, and the smaller openings) must equal the pulmonary return. If this be true then the vena cava inferior returns 5 blood; the other return to the right auricle equals $2\frac{1}{2}$ and the pulmonary return equals $2\frac{1}{2}$. This is not imagination on the part of the writer, for it is made plain: "Das Blut des rechten Ventrikels, je gleiche Teile aus beiden Hohlvenen gemischt, ergiesst sich zur Hälfte durch den kleinen Kreislauf nach links. Das Blut des linken Ventrikels, $\frac{1}{2}$ aus der vena cava inferior. $\frac{1}{2}$ aus den Lungenvenen ergiesst sich zur Hälfte in den Oberkörper, zur Hälfte in die aorta descendens." The aorta descendens therefore receives $\frac{1}{2}$ of the blood from the right and $\frac{1}{2}$ of the blood from the left ventricle = 5.



It will be noted that three things are omitted from the diagram:

1. The coronary circulation which returns through the coronary sinus, but which I suggest as a modification, not a criticism. I take it by "obere Hohlvene" Ziegenspeck implies all orifices other than the vena cava inferior.

2. A certain amount of blood from the descending aorta returns through the superior cava (azygos circulation) and is omitted.

3. The lymph which passes through the walls of the branches of the aorta descendens (probably no small amount) is returned through the thoracic duct to the cava superior.

Returning to the amount of blood in the aorta descendens, if it is equal to 5, then 5—blood must return through the cava inferior (subtracting the amount of azygos and lymphatic return). If each ventricle holds 5 and the blood of the inferior cava is equally distributed, then $2\frac{1}{2}$ + blood is returned by the superior cava (etc.) and the pulmonary veins respectively, but this cannot be the case, for then 5 — blood would pass through the descending aorta. With the major premise that no blood except that passing up the inferior

cava crosses to the left side, the scheme is rendered quite impossible.

The further statement of Ziegenspeck's: "Dass die linke Mündung der vena cava inferior enger ist als die rechte, ändert daran nichts," may be convincing to him, but it hardly covers the physics of the proposition, even if we grant the septal prominence actually does divide the orifice as stated.

The third theory is that of Harvey (1628), who believed that the blood of the superior and inferior cavæ mixed in the right auricle and that mixed blood passed through the foramen ovale. The theory appears quite logical if we but forget the time-honored idea that the head must receive a more arterial blood supply and the relations as they are pictured in the frog and the turtle.

It became evident that experiments must be carried on intra vitam in the embryo to eliminate all danger of artifact, and in consequence the pig embryo was chosen. The writer expresses his indebtedness to Kingan & Co., of Indianapolis, for the many courtesies extended.

The points outlined for investigation were the following:

1. Do both ventricles expel the same amount of blood?

2. In what proportion does the vena cava inferior return blood to each ventricle?

3. In what proportion does the vena cava superior return blood to each ventricle?

4. In what proportion does the sinus coronarius return blood to each ventricle?

5. In what proportion does the blood entering the lungs through the pulmonary artery return through the bronchial veins and pulmonary veins?

6. In what proportion does the blood entering the bronchial arteries return through the bronchial and pulmonary veins?

7. What is the proportion of blood returned to the left heart by the pulmonary veins?

8. In the frog does the blood from the two auricles remain separated in the common ventricle?

9. In the turtle does any of the blood entering the left auricle pass into the cavum pulmonale?

Owing to lack of facilities, question 1, 2, and 3 only will be answered definitely. Much research remains on the problem, and the writer takes the liberty to present this preliminary account because it throws light upon the crossing of the blood currents within the fetal heart. The manner in which the writer intends to obtain the desired results will, however, be touched upon.

The technique in a few words was this: Injection of the selected vein with a suspension of corn-starch granules in normal salt solution; recovery of the blood from the beating heart, both ventricles at the same time and under identical conditions; decolorization of equally diluted specimens and comparison of the two bloods for the number of starch granules. The details of technique are far more complicated to describe than the report warrants.

1. Do both ventricles expel the same amount of blood? This was assumed because of the adult relations, and while there were no grounds for believing otherwise, it was considered essential to substantiate the assumption. A ligature was slipped around the heart at the auriculo-ventricular groove and the ventricles together with their orifices tied off at the completion of ventricular diastole. This was accomplished successfully in two out of ten trials. Each ventricle was bled into a vial and the two sides compared. In both cases the blood was the same, and the question was considered as settled in the affirmative.

2. In what proportion does the vena cava inferior return blood to each ventricle?

Pigs nearly at term were left attached to the placenta and detailed precautions taken to eliminate false circulatory conditions; a quantity of cornstarch-salt solution mixture was injected into the umbilical vein with a hypodermic syringe. An incision was made into the chest, and the heart exposed, and both ventricles stabbed at the same time with identical pipettes. Pigs were rejected if any appreciable hemorrhage occurred or if the heart did not beat at least five times after the pipettes were introduced, or if the blood did not mount equally in both pipettes. Each paired sample was treated alike with ½ of 1% acetic acid to decolorize the red cells. The mortality was naturally great, but 25 paired samples were obtained of which eight were rejected, owing to flaw in dilution. The seventeen remaining paired samples were handled a pair at a time, as follows: each vial was shaken an equal number of times and a given quantity withdrawn and placed under cover. Comparison of the two sides resulted as follows: five were negative (no starch granules) and twelve positive (starch granules). In all twelve the number of starch granules appeared about the same, *i. e.*, they were so nearly equal that it was impossible to determine right from left except from the labels. This experiment proves beyond a doubt that Sabatier's theory is entirely incorrect, and substantiates the Wolff proposition or the Harvey theory.

To prove or disprove the one or the other, a second experiment was undertaken. If starch granules injected into the superior cava were recovered only from the right side, then the Wolff theory was correct and Harvey wrong. However, if the granules were found on both sides then Wolff was wrong and Harvey correct.

3. In what proportion does the vena cava superior return blood to either ventricle? The technique in this experiment was the same as above and would appeal to the reader as relatively simple. It was found extremely difficult to open the chest the full length without injury to the veins at the cervical limit of the thorax. Similarly it was difficult to inject the superior cava high up and inject only a drop or two to avoid undue pressure. Seven samples were finally obtained and submitted to the same requirements and tests. In the seven samples one proved negative (no starch granules) and six positive (starch granules). In four positive the granules were equal on both sides; in one more on the left than on the right, and one (labels unfortunately lost) more on one side than on the other. In both cases the excess

was easily 50%, and was attributed to some flaw in injection or in the recovery of the blood.

The point, however, was settled in the six positive tests. Starch granules injected into the superior cava did pass the foramen ovale, and apparently about equal amounts were found on the right and left side, thus proving conclusively that Harvey's teaching was right, that the blood of the two cavæ mixes in the right auricle and that mixed blood enters the foramen ovale, and showing that if Ziegenspeck, Preyer, and Wolff did find a double orifice to the cava inferior, it in no way inhibits a mixing of the two bloods.

The writer was surprised at the small amount of pulmonary return evidenced in that the starch granules were about equal on both sides, but attributes this to the collapse of the lungs and consequent increase of the capillary resistance and to pressure occasioned on the pulmonary veins by manipulation of the heart. This note will show the difficulties in the remainder of the problem.

The results show that all the organs of the body receive about the same quality of blood, and that the quantity is certainly not the rule laid down by Ziegenspeck. The diagram resulting from this investigation is quite evident, and when published will include the coronary, bronchial, azygos circulations and the lymphatic return.

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TWO CASES OF TYPHOID FEVER WITH INTERESTING BLOOD CRISES.

By Charles P. Emerson, M. D.,

Resident Physician, The Johns Hopkins Hospital; Associate in Medicine, The Johns Hopkins University.

Case I. *Summary.*—A case of typhoid fever, admitted on the 17th, and with death on the 30th day of disease; blood crisis; a period of hypothermia associated with numerous small intestinal hæmorrhages.

W. C., colored, aged 27 years, a fireman, was admitted to Ward M of the Johns Hopkins Hospital October 6, and died October 19, 1906. General number 56,517.

He complained of "dumb chills, loss of sleep, headache, and feeling out of sorts."

The family history was perfectly negative. His parents, four of five brothers, and eight sisters were all alive and well. One brother died when a baby.

Past history.—When a child he had measles, mumps, chicken pox, and whooping cough. He never had had diptheria, scarlet fever, chorea, any form of rheumatism, typhoid fever, pneumonia, or pleurisy. He was once treated for chills and fever, but had never been ill in bed before the present illness. He denied all symptoms suggesting eye troubles; he had a discharge from one ear when a child. He was not subject to colds and had no gastro-intestinal symptoms. He had had gonorrhoea and evidently lues also. He denied having used tobacco and claimed to have always been very temperate in the use of alcohol. His work was not heavy.

The present illness began September 16, on which day he awoke with a headache and general malaise. Although this continued, he worked until September 19. During this time he was quite constipated, and took considerable medicine to relieve this symptom. On September 19 he had to stop work because of the malaise and a very severe headache. That afternoon he had a "dumb chill." These "dumb chills" occurred every afternoon for several days. He described them as periods during which he felt cold. They were not shaking chills. He remained in bed for the next few days, feeling very ill, with poor appetite, headache, weakness, insomnia, and later diarrhœa. On the night of September 29 he awoke in a hot fever, which was followed by a drenching sweat. The next day he came to the Outpatient Department, and quinine was prescribed although on careful examination no malarial parasites were found in the blood. He had no more chills, but he grew progressively weaker, could not sleep, and had a severe diarrhœa of seven or eight movements a day, but had absolutely no pain in the body and no tenderness in the abdomen. He often belched, but had no nausea and no vomiting. He was admitted to the ward October 6, probably the seventeenth day of his illness.

Physical examination.—The patient was a well built, well nourished negro. He lay comfortably on his back in bed. The scleræ were a little yellow, otherwise the eyes were negative. The ears and throat were both negative. The mucosæ were rather pale, the fongue was covered by a thin white fur.

There was general glandular enlargement; the glands of the posterior triangles of the neck, of the axillæ, of both inguinal regions, and the epitrochlears were all enlarged.

The lungs were negative on auscultation and percussion. The heart impulse was in the fourth and fifth spaces, maximum in the fourth, 7 cm. from the mid-sternal line. There was no thrill at apex or base. The deep cardiac dullness extended 10 cm. to the left in the fourth, 2.5 cm. to the right in the third interspace, and above to the lower border of the second rib. The heart sounds were clear, with a relative accentuation of the aortic second sound.

The abdomen was natural in appearance and its movements free. There was some gurgling in the right iliac fossa. No masses were felt, there was no muscle rigidity and no pain on deep palpation.

The relative hepatic dullness began above at the sixth rib in the right mammary line and extended just to the costal margin. Its margin was not palpable. The firm margin of the spleen was easily palpated. The genitalia were negative. There was no œdema of the ankles. The deep reflexes were normal.

The pulse was 80 to the minute, small in volume, of low tension, regular in force and rhythm. The arterial wall was not especially thickened.

Blood examination.—Red cells, 3,896,000; leucocytes, 6800. The red cells were of quite uniform diameter, and of rather pale color. No malarial parasites were found, and no pigmented leucocytes.

Polymorphonuclear neutrophiles	88.8%
Small mononuclears	6.4
Large mononuclears	1.4
Transitionals	2.2
Unclassified	1.2

On October 8, the leucocytes were 4, 620.

October 10. Blood examination: Red cells, 3,752,000; hæmoglobin, 55 per cent; leucocytes, 4,200.

October 11. This morning the stool following a soap-suds enema was pinkish in color and contained three or four small blood clots. The total amount of blood could not have exceeded 15 ccm. This slight hæmorrhage was followed by no symptoms. The temperature, however, dropped from 103° at 4 a. m. to 101.2° at 6 a. m., but in two hours had risen again to 103°. The pulse rose slightly (from 92 to 104) during this drop of temperature, but at 8 a. m. was again 94. Twelve hours later the stools consisted mainly of fluid blood with a few clots, about 200 ccm. in all. We suspected this to be a portion of the same hæmorrhage evacuated earlier.

October 12. A sudden abdominal pain led us to fear that a perforation might have occurred, hence the leucocytes were to-day frequently counted.

10.30 a. m.	11,800
1.15 p. m.	10,300
2.15 p. m.	11,900
3.25 p. m.	13,900
7 p. m.	13,500

At 2 p. m. a third small hæmorrhage was evacuated, consisting of about 200 ccm. of dark fluid and a few small clots.

The temperature on the day before at 8 a. m. was 103°. It fell steadily, reaching 96° at 6 a. m. on this day, and varied from 96° to 98° until 12 m. three days later, October 15, when it rose again. The pulse during that time varied from 94 to 130 and the respirations from 24 to 32.

October 13. The patient looked very ill. He lay with his mouth open and limbs tremulous; the tongue was protruded with difficulty. The pulse was dicrotic and averaged about 112. There was marked pallor of the mucosæ. The abdomen was rather full and rigid, but there was no point of tenderness on pressure. The heart sounds were clear and of normal relative intensity.

Blood examination.—The red cell count was 2,104,000. The stained specimens showed some poikilocytosis and a good many microcytes. Many red cells were polychromatophilic. Hæmoglobin, 30 per cent; color index, 0.7; leucocytes, 16,940. (This includes the nuclei of about 65 nucleated reds.)

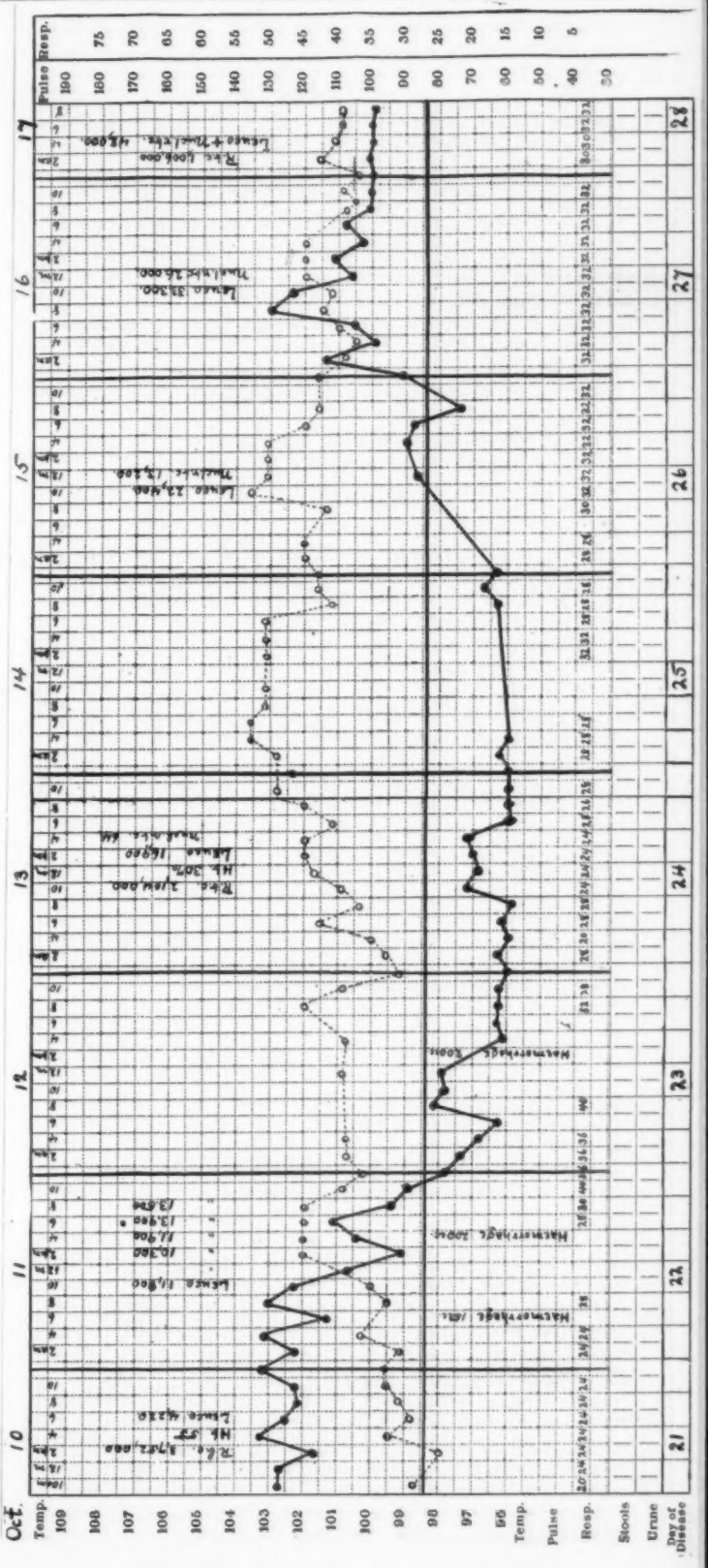
Polymorphonuclear neutrophiles	76.8%
Small mononuclears	16.4
Large mononuclears	3
Transitionals	3
Myelocytes (neutrophiles)	0.2
Unclassified	0.6

No eosinophiles and no mastzellen.

While counting 500 leucocytes two normoblasts were found.

October 15, 9 a. m. The patient looked more ill. The tongue was dry and glazed, there was considerable tremor of the lower jaw, and some subsultus of the arms. The abdomen now was retracted to a remarkable degree. It was rather rigid, but not tender on palpation. The pulse was 124 to the minute, the heart sounds were clear with the aortic second loud. The leucocytes were 30,700 at 2.30 p. m. Temperature subnormal.

4.45 p. m. The patient was very dull and drowsy. He denied that he had any pain. The neck was not stiff. There was no nausea or vomiting, no pain on defecation or micturition. The lungs and heart were both negative. The abdomen was a little fuller than in the morning yet was by no means distended. The



abdominal muscles were held rather stiffly, but there was no local tenderness, no muscle rigidity, or muscle spasm. There was no dullness in the flanks. The liver dullness just reached the costal margin. The lower zone of the abdomen moved on deep breathing very little. Rectal examination was fairly negative, although pressure toward the right caused more pain than toward the left.

The leucocyte count at 5.30 p. m. was 34,600, and at 8.30 p. m. was 37,400.

9.45 p. m. The patient's condition seemed worse. There was more tenderness on the left side of the abdomen and a few suspicious dry râles under the left costal margin. The facial expression was somewhat drawn. The pulse was better than this morning, the aortic second sound was still clear. A typhoid perforation was suspected, and an exploratory laparotomy strongly urged.

10 p. m. Prior to transferring the patient to the surgeons a complete examination was made. This we copy in full.

"On October 11 the temperature fell from 103° to 96.2°. Since then, until this evening, it has not been above 98°, and seldom even 97°. On October 11 the pulse rose from 98 to 120 and since then has not been below 100. This evening the temperature has risen to 99.9° and the pulse to 132. Since October 11 the respirations have varied from 24 to 28; this evening they are 32 per minute. Since admission he has markedly emaciated, is very weak, often irrational, and now is held by a restraining sheet. His arms are twitching. The hands and feet are very cold. The pupils are almost pin-point in size. They react slightly to light. There is no stiffness of the neck. The interior of the mouth is dry, there are sordes on the teeth. The alae nasi do not dilate on inspiration. The posterior cervical, the axillary, the epitrochlear and the inguinal lymph glands are all enlarged and firm.

"The pulse is 142, regular in force and rhythm, small in volume, and low in tension. There is marked arterio-sclerosis.

"*Chest.*—The right front and axilla are hyper-resonant on percussion, and negative on auscultation. The left front is less hyper-resonant than the right. At the left base behind the breath sounds are scarcely heard. On deep breathing they are slightly bronchial in character. The voice sounds at the base are not nasal; the vocal fremitus is there easily felt. The heart sounds are best heard under the left nipple, are clear at apex and base; the pulmonic second is accentuated.

"The abdomen is scaphoid; the respiratory movements are absent below the navel, even on deep respiration. Shadows of loops of bowel are distinctly seen to descend on inspiration.

"*Liver.*—The relative hepatic dullness begins at the lower border of the sixth rib and extends to just below the costal margin. The edge of the liver is just felt. The spleen is not felt, nor is the splenic dullness increased. The abdomen is everywhere soft. There is no local muscle rigidity, no muscle spasm, only a slight tenderness on palpation over the lower extremity of the right rectus muscle, and this is inconstant. In the right iliac fossa is distinctly felt a tumor suggesting a mass of lymph glands at the base of the cæcum. The sigmoid flexure can be rolled under the finger. There is tympany in the flanks.

"*Extremities.*—The shins are clear; there is no œdema of the ankles, no paralysis of the leg and no Kernig's sign. The knee kicks are active; there is patellar clonus. On plantar stimulation there is flexion of the great toes. The deep reflexes of the upper arms are active and there is also marked myoedema. The patient has had no nausea, no vomiting, or hiccough. The bowels have not moved since the last hæmorrhage on October 12. The total of the hæmorrhages has certainly been less than 500 ccm. of blood. There is no tenderness in any joint and no local swelling of the extremities. The Widal reaction tried to-night is very suggestive, but not positive.

"*Blood.*—The leucocytes have risen from 4000 on October 10 to 38,000 this evening. The fresh blood specimen shows marked anæmia. The red blood cells must be very susceptible to injury, since most of them are crenated or otherwise injured, and yet the technique used was careful. A remarkable feature is the large number of nucleated reds, especially megaloblasts and intermediate forms. There are many normoblasts. Many of the nucleated reds have fragmented nuclei. The red blood cells vary markedly in size, but none have a sausage or battledore shape. The majority of the leucocytes are finely granular polymorphonuclears. One typical finely granular myelocyte is seen. An approximate leucocyte count made with the high power showed that many of the nucleated cells counted as leucocytes were red cells and some of them typical megaloblasts. The condition simulates a primary anæmia, perhaps an acute leukæmia with a blood crisis. If the case is typhoid fever, the patient is having a crisis of a most unusual character. Operation not advised."

A most careful study was then made of smears taken during this examination. The total count of nucleated cells was 35,600 per cmm. While making a differential count of 511 leucocytes, there were found 90 normoblasts, 37 intermediates, 9 megaloblasts, and 4 free nuclei. The leucocyte count must have been, therefore, about 28,000, and that of the nucleated reds 7600 per cmm.

Differential count:[1]

Polymorphonuclear neutrophiles	89.43%
Myelocytes	0.97
Small mononuclears	6.26
Large mononuclears	1.18
Transitionals	0.97
Unclassified	1.18

October 16. The patient appeared better and responded well to questions. The alae nasi dilated with inspiration. Respirations were quick, 32 to the minute. The pulse was dicrotic and 110. The temperature had risen to 103°. The mucous membranes were very pale; that of the throat was white and parchment-like, covered with dried secretion. The tonsils were not enlarged. The lungs were negative. The abdomen was still scaphoid, and scarcely moved below the umbilicus. Coils of intestine in active peristalsis were seen around the umbilicus. There was no muscular rigidity and no pain on deep palpation. The flanks were tympanitic. The liver dullness reached just to the costal margin.

The leucocyte count was 59,300, but of these only about 33,300 were white cells, the rest were the nuclei of red cells, for in counting 520 leucocytes there were found, of perfect normoblasts 103; normoblasts with fragmented nuclei 50; microblasts 1; intermediates 30; megaloblasts 15.

Polymorphonuclear neutrophiles	82.3%
Myelocytes	6.46
Small mononuclears	7.79
Large mononuclears	2.28
Unclassified	1.14

No eosinophiles and no mastzellen.

October 17. The patient was distinctly better. The pulse was 104 to the minute, soft and better in quality. The pulmonic second sound was louder than the aortic.

Red corpuscles	1,006,000
Leucocytes	48,000

October 18. The patient now in a muttering delirium.

October 19. Leucocytes 11,340.

[1] All of these counts we owe to Dr. W. D. Gatch, then a clinical clerk on this ward. Ehrlich's triple stain used.

Polymorphonuclear neutrophiles	89.4%
Small mononuclears	9.4
Large mononuclears	0.2
Transitionals	0.2
Unclassified	0.8

While counting 500 cells, 40 normoblasts and 3 intermediates were found.

October 19. The patient died to-day at 4.30 p. m., evidently of exhaustion. He had emaciated extremely.

A trace of albumin was constantly present in the urine, and a few hyaline and granular casts on some examinations. The Diazo was negative. The stools were negative.

Autopsy, No. 2781, October 20, 1906. Fourteen hours post-mortem.

Anatomical diagnosis.—Typhoid fever; ulceration of ileum; acute splenic tumor with anæmic infarctions, and acute perisplenitis; red bone marrow; necrosis of liver and marrow.

(The autopsy notes given here are considerably condensed from the original.)

The body is that of an emaciated negro man, 176 cm. in length. Decubitus ulcer over sacrum, and ulcer on skin of scrotum. The peritoneal cavity is drier than normal, its surfaces are glistening. The intestine is markedly collapsed, and its coils lie away from the spinal column, so that the anterior abdominal wall rests against the back-bone. The spleen is somewhat enlarged; the liver extends below the costal margin. The pleural cavities are free from fluid and adhesions. The pericardium is smooth; its cavity contains no excess of fluid. The heart is not enlarged. The endocardium, valves, and muscular wall are normal. The lungs are rather moist and a good deal of frothy liquid can be squeezed from them, but otherwise they are negative. There is no consolidation anywhere. The spleen weighs 210 gm., and measures 12 x 10 x 5 cm. The surface is rendered uneven by irregular firm projecting nodules, varying in size from 2 mm. to 3 cm. These areas are elevated and quite firm. They are yellowish-white, some with white dots and streaks, and are outlined by a red halo. The remainder of the spleen is fairly firm, and is of a dark grayish-red color. The whole of the spleen is covered by a thin layer of fibrin which is perhaps thickest over the projecting areas, and can readily be removed. On section the firm nodules are seen to be dry, elevated above the cut surface and yellowish-red in color. They are opaque, dull, and dry looking, and show dimly the architecture of the spleen. They are roughly quadrilateral on section and the reddish-yellow central portion is outlined by an opaque, pale yellow line, which is further separated from the remaining splenic pulp by a red line. These nodules are all superficial and extend to the surface. The splenic pulp in general is grayish-red and swollen. The Malpighian bodies and trabeculæ are fairly well seen. In the neighborhood of the opaque yellow areas there are other irregular patches which look dull and dry, and are slightly firmer and red of only a slightly paler hue than the rest.

The stomach, pancreas, duodenum and gall bladder showed nothing worthy of note.

Intestines.—The ileum throughout its upper part is normal looking. There are some Peyer's patches high up which seem swollen and have the appearance of the elevated Chinese letters seen on a Chinese coin. The elevation is in streaks only. Lower, the Peyer's patches are thickened, but not to a very great extent, and rather smooth looking. Near the ileo-cæcal valve there are several ulcers, which vary in size from a few millimeters to almost two centimeters in diameter. Their edges are rather precipitous, but they are not deeply congested. One near the ileo-cæcal valve is about 8 mm. in diameter and is very deep, laying bare the muscle layer. A slight thickening is visible on the serosa opposite. The other patches in the neighborhood are swollen, but not definitely ulcerated. In all there are only three or four small ulcers. The mucosa of the colon is normal looking. The mesenteric glands opposite the ileum are enlarged, pale yellowish in color, and rather firm. The retroperitoneal glands are not enlarged. No definite hæmolymph glands could be recognized. The bladder, rectum, genital organs, and thoracic duct presented nothing of importance.

The kidneys are of about normal size, and weigh 300 gm. The cortex is 7 mm. in thickness. They appear normal on the surface and on cross-section.

The adrenals, aorta, thyroid, and parathyroids all appear normal.

The liver is of normal size. Its surface and cross-section appear normal. No focal necroses were seen macroscopically.

On microscopical examination, the heart showed nothing worthy of note.

The lymph glands in the mesentery show some sclerosis. There are a few large phagocytic cells in the sinuses. There are numerous colonies of bacteria, apparently bacilli, scattered in the tissue. These are also seen in the intestinal wall.

Spleen.—There are well defined anæmic infarcts of characteristic histological structure. In these necrotic areas there are also masses of bacteria. The spleen in general is rather hyperæmic. The venules seem to be filled with a homogeneous hyaline material which stains a grayish-pink and which makes the splenic substance quite solid in places. In other patches the venules contain blood. There are a very few cells containing red corpuscles.

Liver.—There are many definite focal necroses in which the liver cells are lost, and the invasion of polymorphonuclears and mononuclear cells has taken place. Often the outline of the dead liver cell can be made out. The necroses are scattered throughout the lobule, frequently appearing in the middle zone. At the surface of the liver there is one large necrotic area into which no leucocytes have as yet wandered. The capillaries here seem filled with hyaline thrombi.

Marrow of femur.—Sections of bone marrow taken from the femur, and hardened in Zenker's fluid, show a patchy hyperplasia of the marrow cells. Some areas show fat cells only, but in such areas the lymph spaces are widely dilated, and full of a finely granular, pink-staining coagulum. The capillaries throughout are dilated. Areas of focal necrosis are seen, but they are of small size and their fibrin does not take a very deep eosin stain. These areas are found among the fat cells as frequently as in the patches of hyperplasia. The striking feature of the cellular marrow is the great number of nucleated cells, both normoblasts, megaloblasts and intermediate forms. These cells occur as a rule in clumps or islands (Bunting) which are very striking. Some of these islands show as many as 15 or 25 nucleated red cells crowded rather closely together and in the center of such islands one may see a few indifferent marrow cells. The nucleated red cells are less conspicuous in the capillaries and their nuclei smaller and more pyknotic.

Non-granular mononuclears are not numerous and are quite inconspicuous, less numerous even than the nucleated red cells. The small mononuclears occur in small clumps, but none of the large lymphatic nodules described by Longcope are made out.

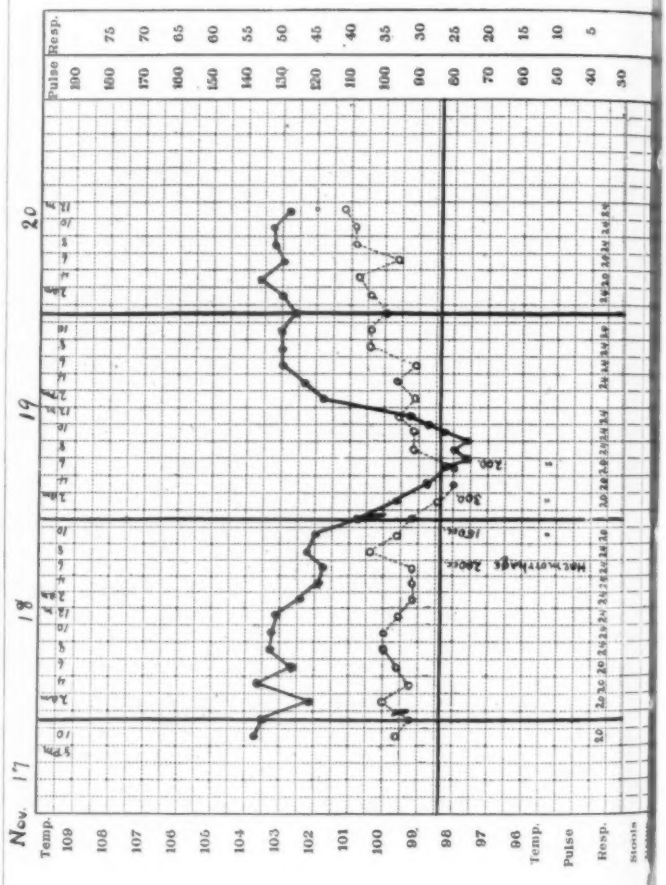
Myelocytes are very conspicuous, being the predominant cells, but the polymorphonuclear leucocytes are relatively few, and evidently most of them have been swept out of the marrow.

Eosinophile myelocytes and leucocytes are easily found. Not infrequently they are found in clumps of from 3 to 9 cells. The bone marrow giant cells are found in small numbers. They are usually of small size, shrunken, and only occasionally show pseudopodia. Large phagocytic cells, situated usually in the capillaries, are found only after prolonged search. They are large mononuclear cells with deeply eosin-staining granular protoplasm, which may contain red blood cells or nuclear fragments. No phagocytes containing coarsely granular yellow pigment were seen.

Cultures made from the spleen, splenic infarct, femur marrow, and vertebra marrow gave pure cultures of Bacillus typhosus. That from the rib marrow, Bacillus typhosus and Bacillus lactis ærogenes.

Our case was, then, one of typhoid fever admitted on the 17th day and with death on the 30th day of the disease. Clinically it was quite characteristic, but the positive diagnosis of typhoid fever was not made until autopsy. Four Widal agglutination tests were reported negative according to our standard, which is a very rigid one, since it demands good agglutination and practically complete cessation of motility in a one-to-fifty dilution, and at the end of one hour. The agglutination at this dilution was good in all four tests, but in each there remained a few motile bacilli. At autopsy four small ulcers of Peyer's patches, were found, which on microscopic examination were typhoid in character. Bacillus typhosus, however, was cultivated from the organs, and this settled the question.

One feature of this case which especially interested us was the period of hypothermia which occurred in association with a few very small intestinal hæmorrhages. On the 22d day of the disease the temperature was 103° F. It began to fall, reached on the following day 96.2° F., and remained at about 96° F. for three full days. Preceding and during the first thirty-two hours of this fall there were passed three stools, which possibly contained 500 ccm. of blood altogether, but we think this is too high an estimate. At the same time the pulse rose from a mean of 95 to a mean of about 120. For the temperature to fall during a hæmorrhage is not uncommon, and yet in a majority of cases, if the temperature chart be shown to a doctor, he cannot pick out the day of the hæmorrhage. In a young, strong patient a sudden hæmorrhage of about one litre of blood is often accompanied by a drop of temperature to a subnormal point. If this hæmorrhage be rapidly evacuated the temperature returns promptly to its previous high point, reaching it in roughly twenty-four hours from the time it began to fall. Contrary to the usual text-book statement, the pulse often does not change at all, but remains low, and even may drop, as in the case of a patient in the hospital at about the same time, a portion of whose temperature curve we give on Chart II. The rate of respiration also shows no change. The delirium often disappears and the patient is for a time mentally much clearer. Sometimes this clearing of the sensorium is accompanied by a very definite feeling of apprehension. There is no pain, no local sensations whatever, but suddenly the patient becomes mentally clearer and is afraid something dreadful is about to occur, but what he does not know. The temperature begins to fall. Then one may be on the lookout for a stool or stools containing blood in large amounts. The apparent temporary improvement in the case explains beautifully the logic of bleeding fever patients, so much in vogue a few decades ago. What more can one ask as the result of a therapeutic measure than that even for one day a delirious fever patient become mentally clear, his temperature fall to normal and the pulse not increase in frequency.



But the case we here report belongs to a different, and not rare class (Chart I). The temperature falls to a low point and remains there for some time. The pulse may rise and the patient may appear even more toxic than before. And yet the haemorrhages are small, often multiple, with the aggregate amount of blood lost much less than in the cases of the former group.

We do not know how to explain the differences. It may be the patient is very weak, emaciated, and almost at the limit of his strength. Such was the case of this patient, but not of another patient in the ward that same season. This was a strong young man, twenty-three years of age. The temperature was already beginning to approach the normal line, when, on the twenty-seventh day of his fever, he had an intestinal hæmorrhage of not over 200 cc. of blood. A period of hypothermia of thirty-six hours followed. It may be a feature of hæmorrhages late in the disease, and after defervescence has begun. It may be that in the case of one sudden profuse hæmorrhage the blood is passed at once, and so there is much less reabsorption of the blood and its decomposition products than when there are multiple small hæmorrhages which remain in the bowels much longer. That is, the whole picture may be more a toxæmia than the result of the loss of so much blood, and of whatever toxins it may contain.

The feature of this case to which we wish to call especial attention is the remarkable blood crisis which occurred during and after this period of hypothermia.

On the 21st day of the disease the leucocyte count was 4,200. On the 22d day the temperature began to fall. On this day the leucocytes were not counted. On the 23d day, with the temperature subnormal, the counts were 11,800; three hours later 10,300; one hour later 11,900; in another hour 13,900; and four hours later 13,500; all practically the same count. On the following day, the count was 16,900. On the 20th day no count. On the following day they had risen; 30,700, 34,600, 37,400, and 35,600 being the counts made at regular intervals. We know, however, that the count 35,600 included the nuclei of 13,200 red cells, and so the white count was 22,400. Without doubt the other counts need a like correction. During the hours of these counts, the temperature was rising. On the 27th day, with the temperature 102.4° F., the leucocyte count was 59,300. Of these cells, however, only about 33,300 were leucocytes, and the remainder were the nuclei of red blood cells. The following day the count was 48,000. The next count was made on the 30th day of the disease, four hours before death. It was 11,300, of which about 10,400 were white, and 900 the nuclei of red cells. Parallel with this rise of the white cells from 4200 to 33,300 and the fall to 10,400, the nucleated reds appeared and rose to at least 26,000, then fell to about 900 per cubic millimeter. Of the 7,600 nucleated reds present on the 26th day of the disease, at least 40 per cent were megaloblasts and intermediates; of the 26,000 present the following day, about 22 per cent were these cells. It was, therefore, a megaloblastic blood crisis. During this crisis, often interpreted as unusual activity of the bone marrow, there was a polymorphonuclear neutrophile leucocytosis, with also 6 per cent of neutrophile myelcocytes, but the eosinophiles were conspicuous by their absence.

We have given elsewhere[2] our opinion of blood crises. We believe that while they are splendid evidence of the struggle the bone marrow is making, the chances are they will not accomplish much in raising the red blood count if the marrow is reduced to the necessity of pouring the larger nucleated cells into the blood stream. This case is a good illustration of this point, for the count on admission was 3,896,000, and four days later, on the 21st day of the disease, it was 3,752,000. On the 24th day it was 2,104,000; and on the 28th day, 1,006,000. That is, the crisis occurred during a rapidly developing anæmia.

It is impossible to give the reason for this crisis in the present state of our knowledge. That it bore a direct relation to the hæmorrhages we do not believe, since it began before and continued after these. We are tempted to bring it into relation with the infection of the bone marrow, for at autopsy a pure culture of Bacillus typhosus was obtained from this tissue, and histologically the bone marrow showed widespread necrotic lesions. This theory is tempting, and is not new, for some cases called acute leukæmia are thus explained. These are cases of rapidly fatal acute infections, with a marked anæmia and a leucocytosis, polymorphonuclear in character, yet with a fairly high percentage of mononuclear cells, both granular and non-granular. From the bone marrow of a few of these cases, pure cultures of pathogenic bacteria have been obtained.[3] It is tempting to regard this case as one of typhoid fever with especial localization of the infection in the bone marrow, causing widespread death of this tissue, a resulting progressive anæmia, and a very vigorous, yet futile effort of the remaining functioning tissue, as shown by the blood crisis, to stem the ebb tide. But how seldom are cultures made from the bone marrow of persons who have died from typhoid fever, and how very seldom is a systematic histological study made of this tissue.

Case II. *Summary.*—A case of typhoid fever with death during the second week. Blood crisis.

E. H., 47 years of age, a fireman, was admitted to Ward E of the Johns Hopkins Hospital September 7, and died September 10, 1907. General number, 60,667.

He complained of "hæmorrhages and weakness."

His family history was quite negative. His past history was also quite negative. He had always been strong and well. Only once had he been confined to his bed, and that was years before, when he had some form of "rheumatism." He never had typhoid fever, pneumonia, pleurisy, or malaria, and denied all children's diseases. He was slightly jaundiced ten years ago.

Until two years ago his digestion had been very good, and until the present illness he has never vomited blood, or passed any

[2] Emerson. The Blood in Pernicious Anæmia. Johns Hopkins Hospital Bulletin, 1907, xviii, 51.

[3] Emerson. Acute Leukæmia. Johns Hopkins Hospital Bulletin, 1907, xviii, 71.

dark stools. During the past two years any indiscretion in diet had caused some gastric pain and a diarrhœa, but nothing more. No cardio-respiratory symptoms were complained of. For one year he had noticed slight swelling of the ankles, but no increased frequency of micturition. He denied all symptoms on the part of the special senses. He used tobacco; and alcohol in great moderation, only about a dozen glasses of whisky a year, but never beer. He worked as a locomotive fireman.

The present illness he dated back to September 1, although his friend stated that he had noticed that the patient had looked pale and sick for a week before that. On September 1 he noticed that his stools contained large black blood clots, and since then the stools had been very black and tarry, but had never contained fresh blood. Two days before this he had taken a dose of calomel. On September 1 he also vomited about a cupful of bright red blood, and again, about the same amount September 5.

He had lost strength rapidly during this week.

On admission, September 7, the patient was found to be a stout man, weighing between 200 and 210 pounds. He was lying flat on his back, and complained of no pain or discomfort. His skin and mucous membranes were very pale. The respirations were 28, and the pulse 120 to the minute. The scleræ, pupils, pupillary reflexes, and movements of the eyeballs presented nothing of importance. There was no jaundice.

The throat was negative, the tonsils not enlarged.

There was no glandular enlargement.

The lungs were typically emphysematous.

The heart presented nothing of importance. It was neither hypertrophied nor dilated.

The abdomen was practically normal. The spleen could not be felt. (The patient was stout, and held his abdominal walls rigidly.) The relative hepatic dullness began at the 6th rib and descended to just below the costal margin, but its edge could not be felt (perhaps for the same reason that the spleen was not). There were no dilated veins on the abdomen.

There was slight œdema of the ankles. No purpuric spots were seen anywhere on body.

There were 1,300,000 red blood corpucles, and 85,000 leucocytes per cubic millimeter. Hæmoglobin, 25 per cent (Sahli's hæmometer). The red cells were rather pale, and many of them on staining were polychromatophilic. The blood platelets were very numerous. Differential count. (Ehrlich stain; Dr. Clough.)

In counting 530 leucocytes, 88 normoblasts, 7 intermediates, and three megaloblasts were found. There were, therefore, about 71,500 leucocytes, and 3500 nucleated reds per cubic millimeter.

Polymorphonuclear neutrophiles	74.5%
Neutrophile myelocytes	8.8
Cells intermediate between above two groups	6.3
Large mononuclears	6.2
Small mononuclears	3.3
Eosinophiles	0.2
Mastzellen	0.
Unclassified	0.7

September 8. No apparent change.

September 9. Nucleated blood cells, 90,000 per cmm. No purpuric spots, no enlargement of the lymph glands could be found. The respirations and pulse now become more rapid.

September 10. The patient died early this morning.

The temperature while in the hospital was 99.6° on admission, but rose at once to 103°, and after that the extremes were 101° and 104.4° F. The pulse varied from 100 to 146. The respirations, at first about 28 to the minute, were later from 40 to 44.

He passed while in the hospital about 1200 cc. of urine a day—that passed on admission was clear, but on the day of death it contained a trace of albumin and a few hyaline and finely granular casts. The five stools were tarry.

In blood cultures, made September 9 by Dr. Beall grew later a pure culture of Bacillus typhosus.

Most unfortunately no autopsy was allowed.

This case came to us with the diagnosis of cirrhosis of the liver, rupture of œsophageal varices, and secondary anæmia. We found very little evidence of cirrhosis of the liver, and diagnosed it as one of the cases now often included in the acute leukæmias, meaning by this an infection with this unusual blood picture. The discovery of Bacillus typhosus in the blood leaves no doubt that it was a case of this infection. It is very possible that the man had cirrhosis of the liver, and that the hæmatemesis and melæna were due to a bleeding œsophageal varix. We cannot say. The interest for us lies in the effect of hæmorrhages in typhoid fever on the blood picture. The blood picture of Case II resembled that of Case I. In both there was a severe anæmia, a very high leucocytosis, with a fairly large number of typical neutrophilic myelocytes, very few eosinophilic leucocytes, no mastzellen, and a megaloblastic crisis. The eosinophile cells during typhoid fever are usually very much reduced, and the failure of an increase when there is a high leucocytosis may be of value to those interested in these cells. There was no period of hypothermia while in the hospital, but the hæmorrhages had begun seven days before admission, and the temperature was not taken during that time.

Whether in this case, also, there was a severe local infection of the bone marrow as in Case I, cannot, unfortunately, be known.

These cases illustrate well the fact that very high leucocyte counts should be controlled by differential counts to avoid counting nucleated red corpuscles as leucocytes.

As we have mentioned above, such cases have been reported as examples of acute leukæmia, and in discussing acute leukæmia, features of such cases as these are sometimes given. (This question is discussed and references given in an article referred to above.) But we believe that the high percentage of polymorphonuclear neutrophiles, the relatively low percentage of typical myelocytes, and the very low percentage of eosinophiles, in connection with many nucleated reds, may allow us to keep these cases separated from the leukæmias. We know the cause of a few at least of these cases, while of true leukæmia the cause is still utterly unknown.

It is in the hope of stimulating a little interest in this problem, and because we judge that such a blood picture in typhoid fever is very rare, that the above cases are reported.

MRS. PACKARD AND HER INFLUENCE UPON LAWS FOR THE COMMITMENT OF THE INSANE.[1]

By W. R. DUNTON, JR., M. D.,

Assistant Physician Sheppard and Enoch Pratt Hospital, Towson, Md.

In June, 1906, there came into my possession two volumes, the first entitled "Modern Persecution, or Insane Asylums Unveiled," and the second, "Modern Persecution, or Married Women's Liabilities, as demonstrated by the report of the Investigating Committee of the Legislature of Illinois," by Mrs. E. P. W. Packard, published by the authoress, and printed in Hartford in 1874. These books are bound in cloth stamped to imitate leather, with considerable gilding. The edges are gilt and the make-up of the books is not in keeping with their character. A glance at them shows that they were written by one of that class of chronic insane, who on account of the apparent mildness of their alienation are frequently enabled to cause great trouble to those who are responsible for them, and who oftentimes have been able to publish their experiences and to write them in such a way that they do not impress a person untrained in observing the insane as being abnormal.[2]

Mrs. Packard, I learned, had done so much that a closer study of her works and of the records of the times in which she lived has proved very interesting. Among other things, she is responsible for changing the laws regarding the commitment of the insane in Illinois and in Massachusetts, and I felt that possibly others might be interested in learning something about her.

A brief history is necessary for a proper understanding of her case. According to her own account, her maiden name was Elizabeth Parsons Ware, and she was born December 28, 1816, at Ware, Hampshire County, Mass. She was the only daughter of an orthodox clergyman of the Congregational denomination, and was married May 21, 1839, to the Rev Theophilus Packard, fourteen years her senior, who was a Congregational clergyman preaching in a Presbyterian church in Manteno, Kankakee County, Ill., at the time when what she terms her "legal prosecution" began. She had then been married twenty-one years, and she had borne six children, the oldest eighteen years of age and the youngest eighteen months. She states that she had been educated a strict Calvinist and her reasoning faculties had been developed through scientific education, so that by the simple exercising of her own reason and common sense she had been led to endorse theological views in conflict with her educated belief and with the creed of the church with which she was connected. Knowing that she had some original ideas in regard to religious belief she was invited by a deacon to join his bible class and present her views, with the idea of awakening interest in the study of the bible. The matter was referred to Mr. Packard, who acquiesced, and the patient accordingly presented a paper dealing with the free discussion of religious belief, rights of private judgment, etc., all of which were somewhat at variance with the Calvinist doctrine.

The discussion which was started led to an increase in the size of the class from six to forty-six, but it was felt by the orthodox deacons that Mrs. Packard's views were iconoclastic and that she should not be allowed to promulgate them, and in the following extract we find reference to the first effort made by her husband to stop these discussions.

"One afternoon Deacon Smith visited him [Mr. Packard] in his study, and held a secret interview with him of two hours length, when he left him a different man. That evening, just before retiring to rest, he remarked in a very pleasant tone:

"Wife, I want to talk with you a little while, come here." I went into his extended arms, and sat upon his lap, and encircled his neck with my arm, when he remarked in a very mild tone of voice:

"Now, wife, hadn't you better give up these bible-class discussions? Deacon Smith thinks you had better, and so do some others, and I think you had better too."

"Husband, I should be very glad to get rid of the responsibility if I can do so honorably, but I do not like to yield a natural right to the dictation of bigotry and intolerance, as Deacon Smith demands; but I am willing to say to the class that as Deacon Smith, and Mr. Packard, and others, have expressed a wish that I withdraw my discussions from the class, I do so, at their request, not from my desire to shrink from investigation on my part, but for the sake of peace, as they view it."

"No, wife, that won't do; you must resign yourself."

"Won't that be resigning, and that too on a truthful basis?"

"No, you must tell them it is your choice to give them up."

"But, dear, it is not my choice."

"But you can make it so, under the circumstances."

"Yes, I can make it so, by stating the truth; but I can't by telling a lie."

"Well, you must do it!"

"O husband! how can you yield to such an evil influence? Only think! Here you have pledged before God and man that you will be my protector, until death part us, and now you are tempted to become my persecutor! Do be a man,

[1] Read at a meeting of The Johns Hopkins Hospital Historical Club, March 11, 1907.

[2] Comparatively recently (1904) there was published in Baltimore a book of like character entitled "Sane or Insane? or How I Regained Liberty," by Margaret Starr, this being the pen name of Miss Stella Lawson, whose case occupied the papers for a brief period. It shows a great many similarities to the books under discussion.

and go to the class, in defiance of Deacon Smith, and say to the class: 'My wife has just as good a right to her opinions as you have to yours, and I shall protect her in that right. You need not believe her opinions unless you choose; but she has a right to defend her honest opinions as well as yourselves. I shall not suffer her to be molested in this right.'"

"Then you will be a man—a protector of your wife—and you will deserve honor, and you will have it. But if you become my persecutor and go against me, as Deacon Smith desires, you will deserve dishonor, and you will surely get it. Don't fall into this fatal snare, which the evil one has surely laid for you."

"He construed my earnestness into anger, and thrust me from him, determining to risk this result at all hazards."

"From that fatal time, all good influences seemed to have forsaken him, and he left to pursue a downward way, with no power to resist evil or flee from the tempter. Reason, conscience, judgment, prudence, consistency, and affection, all, all, directly sunk into the fatal sleep of stupidity or death."

"From that point, I have never had a protector in my husband. He has only been my persecutor! In a few weeks from that time, he forcibly entombed me within the massive walls of Jacksonville Asylum prison, to rise no more, if he could prevent it. He told me he did this, to give the impression that I was insane, so that my opinions need not be believed, for, said he, 'I must protect the cause of Christ!'"

Upon Mrs. Packard's refusal to discontinue her "missionary work" her husband wished her to visit her brother for three months, and take two of the children, but refused to allow her to have any money at all, although apparently she had a certain amount in her own right, and at the end of the discussion he threatened to send her to an asylum. She consulted a neighbor, who told her that it would be impossible for her to be incarcerated in an asylum without a jury trial, and that no jury would pronounce her insane. She then details how her children were alienated from her, how people were introduced into her house to spy upon her, how her things were taken from her and finally, how, on June 18, 1860, she was abducted, as she expresses it, and sent to the State asylum at Jacksonville. She states that the only medical examination that she had was when the two physicians came to her room and each felt her pulse without asking her a question or saying a word to her, and that she was incarcerated on a statute of the State of Illinois, passed February 15, 1851, which is as follows:

"Married women and infants, who, in the judgment of the medical superintendent (meaning the superintendent of the Illinois State Hospital for the Insane) are evidently insane or distracted, may be entered or detained in the hospital on the request of the husband of the woman or the guardian of the infant, *without* the evidence of insanity required in other cases."

It can readily be seen how such a statute as this was capable of causing much persecution. On Mrs. Packard's refusal to do anything which could be construed as a voluntary effort on her part to enter the asylum, she was picked up and carried to the train, and from the train to the asylum, where she was received by the assistant superintendent and was conducted to her "lone and solitary cell." The next morning she had quite a long conversation with the superintendent, Dr. McFarland, upon whom she apparently made a favorable impression, and a few days afterwards took leave of her husband, bidding him "good-bye forever."

She apparently got along well for the first four months of her stay at the asylum, and felt that Dr. McFarland was an honorable man. She states, however, that on one occasion he kissed her upon her forehead, and upon her reproving him he stated that it was only a kiss of charity. She was allowed free use of writing materials, and feeling that she had influence over Dr. McFarland she felt that she could induce him to treat the patients in a more humane manner than he did, and with this idea in view she wrote him a reproof, covering eighteen printed pages, in which she made certain threats after beginning in a most religious way. She was then sent to a "disturbed" ward where she remained for two years and eight months, and her accounts of what she did are extremely interesting.

It is difficult to judge the true state of affairs from Mrs. Packard's statements. At times she speaks of the filthy condition of the wards, the neglected condition of the patients, and of the cruelties to which they were subjected, and in another place will state how through slight efforts on her part or through a humane nurse everything was made bright, clean, and cheerful. This inconsistency in statement is a rather prominent feature throughout the book, which is put together in a most slipshod manner. Apparently each chapter was written as a separate essay, and there is consequently a great lack of consecutiveness. The patient wrote a great deal although she was supposed to be deprived of writing materials, but obtained them by various subterfuges, and it is difficult to reconcile her statement that she is a Christian woman with many of the underhand acts of which she was guilty. She was apparently meddlesome, and I imagine must have led the attendants and physicians a rather uncomfortable existence.

She intersperses her narrative with frequent histories of other patients and with condemnation or approbation of various attendants.

In 1861 she made an effort to interest the chaplain in her behalf, created a disturbance, and was carried back to the ward after chapel by a porter. It may perhaps illustrate Mrs. Packard's point of view to state that she met a patient at a ball, and on asking him his name was told that it was Jesus Christ, but that he was not the Jesus Christ who came to earth and was crucified, but another, and that in God's family all his sons were Jesus Christ as much as the first, as soon as they became perfectly developed in his spirit which the patient felt was perfectly developed in himself. In other words the patient felt that he was a perfect man in the estimation of Jesus Christ. Mrs. Packard asks, "Now, where is his insanity? Even his hobby, where has that gone?"

In September, 1862, a few days prior to the meeting of the trustees, she asked the superintendent's permission to read to them a paper directed at Calvinism. Mr. Packard was present on this occasion, and for fifty minutes the patient read "An Exposure of Calvinism and Defense of Christianity," the reading of which created such a good impression that she asked permission to read another document which was entitled "Conspiracy," and was an exposure of Dr. McFarland's and Mr. Packard's actions against her personal liberty.

Following this she was questioned by the Trustees, and she states that "The playful easy style and manner in which I made my statements seemed to dissipate the sentimental gravity of this august body so that they came to seemingly regard me as one of their number instead of a culprit under the grace of court! They manifested a willingness to do anything whatever that I asked." Mrs. Packard called their attention to the fact that her husband was not compelled by law to support her, that she had no legal protection of her person nor of her rights except through the voluntary act of her husband. The next day Dr. McFarland requested that she give him the documents which she had read, and promised that both would be printed. She wished to make certain changes, and ended by writing a book which was called "The Great Drama," of which she states:

"If, during my life-time, this 'Great Drama' can be published and not imperil my personal liberty, I shall be happy to give it to the world."

"But until that time arrives, when an original thought can be spoken or written, without incurring the charge of insanity for such an act, my personal liberty is only safe, while this manuscript is hidden from the age in which it was written."

The paper which she read on Calvinism forms Chapter LIV of her book. Following this occurrence she was treated very well, but after again threatening the superintendent she was again sent to a "disturbed" ward. The superintendent agreed to discharge her, but the trustees decided that she should only be discharged into the custody of her husband. This Mrs. Packard resented, and thereupon she was removed by force from the asylum just as she had been placed there June 18, 1863. This ends the first volume.

In the second volume Mrs. Packard states that she protested against again being placed in the hands of her husband because she knew that he would send her to an asylum in Massachusetts, but despite her protest she was put "into the absolute power of my persecutor against my will." Her husband placed her in the care of her adopted sister in Putnam County, Ill., with instructions that Mrs. Packard should never be allowed to leave, that if she did he would place her in another asylum. She made friends who agreed to stand by her in case of any trouble, and advised her to go to her children, and gave her the necessary money. She arrived at Manteno, her home, and happened to meet her little boy at the station. From him she learned of certain commands that Mr. Packard had made in order to prevent intercourse between her and her children. She was told by her husband that she was not wanted, and that she should not take any part in the household work nor interfere with the children in any way, but despite this, Mrs. Packard went about her household occupations and states that she had made up her mind never to speak in self-defense.

When she found the keys which were used to keep her from the household stores she appropriated them, and following this an active search was made of all her things and even the house, she finally being locked up in a room so that she could have no opportunity of using these keys. The windows of her room were nailed down and her meals were brought to her. She was not allowed to communicate with anyone excepting her own family, but for six weeks she taught her four children arithmetic, grammar, etc., she having at one time been a teacher.

Mr. Packard accidentally left a package of letters in her room from which she learned that in a few days she was to be entered into the "Northampton, Mass., Insane Asylum, for life." She comments upon the fact that the superintendent, Dr. Prince, was willing to receive her as a case of hopeless insanity on a certificate of Dr. McFarland without any sort of a trial, and that Mr. Packard's sister had evidently assisted him in making the arrangements for her transfer.

She also learned that the trustees at Jacksonville were willing to receive her again as a patient. She was able, shortly after, to send a note to her friend, Mrs. Haslet, by means of a passer-by, and a writ of habeas corpus was secured. Mrs. Packard then had a jury trial which continued five days. She reports this trial, which took place in January, 1864, at considerable length, by means of statements of various persons concerned in it. The only ground upon which this trial was secured was the fact that she was kept a prisoner in her house.

Following the trial she found that Mr. Packard had removed in the meantime to Massachusetts, and she had been deprived of her property and was without means of support. In attempting to gain possession of belongings which had been left in the charge of her brother-in-law she committed certain acts which were said to have been illegal and in which the question of her nonentity, because she was a married woman, was brought out somewhat prominently.

In order to support herself Mrs. Packard decided that she would publish her book entitled "The Great Drama, or the Millennial Harbinger," and sold tickets at fifty cents each, by means of which she raised $700.00 and was enabled to have 1000 of these books printed. After she had sold about 6000 books she visited her father in Massachusetts, was forbidden by Mr. Packard to see her children, but succeeded in winning her father to her cause, and he became her protector. She thought it necessary to "seek the protection of the legislature of Massachusetts by petitioning them for a change in their laws on the mode of commitment into insane asylums." She introduced two bills into the Massachusetts legislature, one that "No person shall be regarded or treated as an insane

person, or a monomaniac, simply for the expression of opinions, no matter how absurd these opinions may appear to others," and the other that "No person shall be imprisoned and treated as an insane person except for irregularities of conduct, such as indicate that the individual is so lost to reason as to render him an unaccountable moral agent."

She also succeeded in having the mode of commitment changed so that instead of allowing the husband to enter his wife at his simple request, added to the certificates of two physicians, a form of commitment now in force in Maryland, it was necessary that the husband get ten of the wife's nearest relatives to join in the request for admission to an asylum, and made it possible for the patient to communicate with any of these ten relatives beside two other persons who might be designated. With the passage of this law Mrs. Packard felt somewhat secure.[3]

Mrs. Packard interrupts her narrative here to villify her husband and to explain why she did not get a divorce. As has been stated before, her narrative is not consecutive, and of itself indicates mental instability. She also narrates a dream which she had had while at Jacksonville, and in the interpretation of which she had been assisted by a woman in charge of the sewing-room. She also speaks of having been warned two and a half years before her admission to Jacksonville by spiritualistic communications, of her impending troubles. She speaks of Mr. Packard's monomania and says that three years before his plot culminated that she had visited a lawyer in New York State to obtain advice on the subject of woman's rights, from which it may be inferred that Mrs. Packard had been active in other propaganda than bible teaching. She states that Mr. Packard was examined by Mr. Fowler, the great phrenologist, who expressed his opinion somewhat as follows:

"Mr. Packard, you are losing your mind—your faculties are all dwindling—your mind is fast running out—in a few years you will not even know your own name, unless your tread-mill habits are broken up. Your mind now is only working like an old worn out horse in a tread-mill."

After preparing the ground by obtaining testimonials and making friends, Mrs. Packard left Massachusetts in the winter of 1866 and came to Chicago to organize an effort for the passage of the Personal Liberty Bill of married women. She brought the matter before the governor, whom she enlisted upon her side, and after considerable work on her part, opposed as she believed by opposition organized by Dr. McFarland, she succeeded in having the bill passed.[4] This bill provided that no person should be incarcerated in an asylum for the insane without a jury trial; and that all patients then in such hospitals should be given a jury trial. This bill was approved by the governor March 5, 1867. An investigating committee was also appointed, who reported adversely upon the asylum management and recommended that Dr. McFarland be removed. Mrs. Packard interpellates considerable testimony from attendants and former employees, etc., all derogatory to the hospital management, and gives an account of how Dr. McFarland had "cruelly punished a perfectly sane patient" for striking an attendant, and also how he had made an improper proposal to a sister of one of the patients.

The Board of Trustees, who alone had authority to remove Dr. McFarland, was appointed by the governor, and in order to shield themselves requested that they be re-appointed, agreeing to discharge Dr. McFarland. This they did not do, but re-appointed Dr. McFarland and resigned themselves, thus keeping Dr. McFarland in office for a further period, as no further steps could be taken tending toward his discharge until the next session of the legislature. Dr. McFarland resigned before this met, and in this manner was cleared from any opprobrium which might attach itself to him.

Mrs. Packard next turned her attention to Connecticut, and made an attempt to have the laws for the insane changed in that State, but was not successful.

During the session of the Iowa legislature of 1872 she made a great effort to have a bill protecting the rights of the insane passed and was successful.

The remainder of the book is made up of arguments concerning her past work, narratives of ill-treatment of patients at Bloomingdale, Trenton, and other hospitals, and a number of statements as to her own employment, from which we learn that she settled down in Chicago with her children until they gradually left her to seek new fields of occupation.

From Mrs. Packard's own statement, as contained in these volumes, we have learned that she was instrumental in changing the commitment laws of two States, Massachusetts and Illinois, of securing the passage of a bill to protect the inmates of insane asylums of Iowa, and secure the patient's right of free correspondence, and also of having the superintendent of the Jacksonville Asylum removed from office.

From these same books we also formed the opinion that Mrs. Packard was a chronic lunatic whose judgment was impaired, and who in all probability might be controlled by delusions, and the question of diagnosis is an interesting one although it is impossible to be strictly accurate. She evidently belonged in a rather large group of cases whom Magnan placed under his *délire chronique*, and by others grouped as chronic delusional insanity, in which the symptoms are not especially well defined. Her condition also may be classed either as a menopausal or a conjugal psychosis. At the time of her incarceration in Jacksonville she was 44 years old, about the time when her climacteric might be supposed to have begun and after she had accomplished certain reforms she seems to have settled down to a quiet life, possibly at the expiration of her climacteric and the subsidence of the mental symptoms. The fact that her husband was 14

[3] These bills were presented to Committee of Lunacy, March 29, 1865, and form Chapter 268, Section 2, "Laws of Massachusetts."

[4] In the American Journal of Insanity, Vol. 22, page 548, in a review of the report of the Jacksonville Asylum, for 1864, Dr. McFarland speaks of mild cases of insanity and of the trouble that they may cause, but makes no specific reference to Mrs. Packard.

years her senior may have been an added factor, and while there seems to have been no open rupture between them, preceding his declaring her insane, there may have been some waning of their affection. It has been difficult to find any literature upon the other side of the case, but the following was obtained after a good deal of trouble.

On May 19, 1863, Dr. Andrew McFarland, at the meeting of the American Association of Superintendents of American Institutions for the Insane, held in New York City, read a paper entitled "Minor Mental Maladies," which is printed in the Journal of Insanity, Vol. 20, Page 10, July, 1863, and in the discussion which followed the reading he relates (page 89) a case history, which is evidently that of Mrs. Packard. According to this she was admitted to Worcester Hospital when 20 years of age; her father was a man of sufficient wealth to have given her a superior education, and she was very accomplished; she possessed a fine personal appearance, and was a model of a young lady in respect to her moral, physical, and intellectual qualities. At 19 years of age she was the principal teacher of a first-class Massachusetts female school. Her mother had been insane for many years. About five or six years after her marriage she began to thwart her husband in little matters, but he kept the facts to himself, but matters becoming too troublesome he removed to the State of New York. The same disposition again showed itself and he again removed, going to Ohio, and about 1857-58 they came to Illinois. Dr. McFarland could not, at the time of her admission, discover any intellectual impairment at all, but states that "her hatred of her husband had something diabolical about it; every instinct of love was banished from her. She was thoroughly demoralized and corrupted in all her moral sentiments. . . . She informed me that her husband was the great 'red dragon' and that her eldest son was the 'man-child' mentioned in the same Apocalyptic connection, and that was the only delusion discovered in two and a half years."

Dr. McFarland states that she gave him infinite trouble, and then speaks of her paper on Calvinism to which reference has been made above.

In writing her book "the whole delusion which had lain concealed in her case for eighteen years became fully developed, and it showed that all this perversity of conduct arose out of one single delusion; and the delusion was, that, in the Trinity, distinctions of sex had to exist; that there could be in the Trinity no more than in the family unity of sex; that there must be a distinction of sex, and that she was the Holy Ghost."

From his remarks Dr. McFarland evidently believed the case to be one of moral insanity.

In the *American Journal of Insanity*, Volume XXVI (Oct., 1869), page 204, there is a review of five pamphlets dealing with the Illinois legislation regarding hospitals for the insane which begins as follows:

"For the last two or three years, or more, the State of Illinois has been singularly under the influence and dictation of a handsome and talkative crazy woman, and of a legislature prompted by her to be crazy on at least one point—that of the State Hospital for the Insane—as if they expected some day to inhabit there, and wanted to make of it, in advance of their going, as lawless a place as a legislative hall in these days is commonly reputed to be."

This vein of sarcasm is carried through several pages, and attention is called to the fact that during Mrs. Packard's confinement in the State Hospital "the idea, not very irrational, that the kind and civil superintendent was, in her own phrase, a true man, made in the image of God, and whom she might adore in preference to any 'red dragon'" (her husband), her offer of attachment being unreciprocated, she turned upon Dr. McFarland. The above statement is in measure borne out in her own works.

Criticism is made of the manner in which the committee carried out their investigation of the hospital mismanagement. The fact that but few of the meetings were held at the hospital, that the meetings were not published, that no publication was made of evidence offered, and that the accused persons, excepting only the superintendent, were not given an opportunity of hearing the charges made against them and of making excuses, being the chief points of attack.

The question of the manner of taking evidence is discussed at some length, and also a statement is made of the position of the hospital toward the patient, it being claimed that it stands *in loco parentis*.

Quotation is made from the report of the Trustees as to their manner of carrying out the provisions of the Personal Liberty Bill, from which I quote the following:

"Three sets of cases seemed to grow out of this stage of the transaction:

(1) Those maintaining entire silence.

(2) Those refusing participation in any proceeding.

(3) Those desiring trial.

These latter had their subdivision into those requiring trial at the institution and those wishing it at their homes. The result of these inquiries (made in a manner to give the utmost freedom in reply) will throw some light, hitherto unperceived, on the feelings of the insane. The first class was most numerous of any; the second was next in point of numbers; and the third class least of all; and of those expressing inclination for trial, eleven, only, desired such trial to take place at their homes. On communicating the wish of the latter number to their legal representatives, the refusal was unanimous to be at the expense of such transportation."

Just criticism is made of this manner of committing a sick person to a hospital, and the contention is made that it would be as well to hold a like investigation of other sorts of hospitals.

An example of how detrimental a public jury trial may be for an insane person is given, and in conclusion this backward step by the State of Illinois is deplored.

From the *American Journal of Insanity*, Vol. XXVII (October, 1870), page 260, we learn that Dr. McFarland, the late Superintendent of the Illinois Hospital for Insane, has proposed himself as a candidate for the State legislature of

Illinois; with the hope, if elected, of enlightening the people of that State on their public charities. "A fascinating crazy woman, who managed to seduce partisan prejudice and ignorance for her allies," is referred to as well as the recent legislation, and quotation is made from Dr. McFarland's manifesto upon the subject.

In the same journal, Vol. XIX (October, 1872), page 251, Dr. Ranney, Superintendent of the Iowa Hospital for Insane, at Mt. Pleasant, refers to the recent bill and speaks of Mrs. Packard's agitation, saying that from information he had received, the "legislation had been brought about by the grossest misrepresentations of this person, Mrs. Packard."

In the same journal, Vol. XXX (October, 1873), page 175, in a report of the proceedings of the Association of Medical Superintendents for Asylums of the Insane, Dr. Gray, Superintendent of the Utica State Hospital, states that "There have been some attempts to modify legislation in our State. We have had Dr. McFarland's former patient, Mrs. Packard, in consultation with the legislature. She did not accomplish much, however, in the introduction of Illinois and Iowa Bills, or what she called the 'Personal Liberty Bills'; and further says: 'She had two or three bills there and might have done some mischief, but the fact was too apparent that she had herself a record, and that record is in the proceedings of this Association, in the discussion in New York, a good many years ago.' I was asked in regard to the matter, and replied by handing her case to a member of the committee who had this matter in charge, as given in the *Journal of Insanity*, by Dr. McFarland, some years ago. Also a report of the legislative investigation into the affairs of the State Asylum of Illinois; a part of which proceedings contained the matter of her claiming Dr. McFarland as her affinity, that the doctor was the first person who had fully sounded the depths of her connubial life, that she was the third person in the Trinity, and her son was Jesus Christ. One of the waggish members hearing this, naively remarked 'that he did not want to oppose her, because he would be resisting the Holy Ghost.' She conceives it to be her divine mission to visit all the States, and does not believe in her insanity. I suppose she will soon be up in Massachusetts, at least we commend her to some other State."

In the same journal, Vol. XXXI (October, 1874), page 138, we find the following: "In relation to the subject of legislation we have had a little experience with Mrs. Packard. She is still with us, button-holing legislators, and pressing her bill. After her appearance before the legislative committee on public charitable institutions, it was evident they were satisfied, that her statements were entitled to receive little attention, and also with the unsoundness of the views of those who appeared in her behalf. It is true, however, that this committee, probably from motives of policy, reported to the legislature that it would be expedient to increase to a limited extent, the authority of the Board of State Charities, and directed superintendents to allow patients to write, free from any supervision, to this Board."

The above remarks being made by Dr. Eastman, Superintendent of the Worcester Lunatic Hospital, Worcester, Massachusetts, and on page 151 of the same, Dr. Basset, Superintendent of the Iowa Hospital for Insane, at Mt. Pleasant, refers to the law permitting unrestricted correspondence as follows: "We have now had two years of such legislation, and excepting a few modifications, scarcely lessening the evil which has abundantly developed under the practical working of the law, it still stands upon our statutes, a monument of unwisdom."

He then states that the hospitals have been visited regularly by a visiting committee to whom complaint has been made and who have investigated the same, and says that the above law was changed last winter so as to lessen the amount of letter-writing, so that the patients are now permitted to write under seal only to the visiting committee and only receive letters addressed to them from the committee without inspection, and says: "I have desired to call your attention thus briefly to this matter, for the reason partly that Mrs. Packard, the author of our law, is not idle, and I have reason to know intends to prosecute her efforts in other States. I heard her declare it to be her intention to make this her life work."

I have been unable to learn when Dr. McFarland resigned, but he subsequently established in Jacksonville a private hospital called Oak Lawn. Apparently he was well thought of by his associates. Neither have I been able to learn what finally became of Mrs. Packard, but I hope that later I may be able to settle these points.

Note.—Since the above was read it was learned that Dr. McFarland died at Jacksonville, November 22, 1891. It is understood that Mrs. Packard survived him. One of her sons was associated with Dowie, but after a quarrel with the prophet left the organization. Several of his children have been treated for nervous disturbances.

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